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A FATAL CASE OF TETANUS TREATED WITH TRACHEOTOMY AND HYPOTHERMIA

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The modern management of tetanus has aroused recent interest.[1-4] There are, however, few reports in the English literature on the use of hypothermia in tetanus in children. The following case, despite the unsuccessful outcome, is considered worthy of comment, as an illustration of the present-day approach, and the problems that arise in the treatment of tetanus.

CASE REPORT

A 4½-year-old boy fell down a few steps and sustained an open wound over the occiput, which was treated by the mother. Ten days later, the child fell ill, refusing to open his mouth, and complained of a stiff neck. During the night, he had a convulsion, and was admitted to hospital the following morning.

On admission the classical features of tetanus were present, viz. trismus, meningismus and opisthotonus, with a septic wound over the occiput. The patient was fully conscious. Soon after admission a tetanic convulsion occurred. For the 1st day treatment consisted of sedation, nursing in a darkened room, intravenous fluids and large doses of antitoxin and penicillin. The following day the patient had a severe convulsion with pharyngospasm, cessation of breathing, cyanosis, and temporary loss of consciousness. As these convulsions were repeated and swallowing became impossible, tracheotomy was performed, gastric contents were sucked out, the wound was excised, further sedation was given, and fluids were administered intravenously.

To induce hypothermia, treatment with Largactil (chlorpromazine) was commenced on the 2nd hospital day, with an intravenous dose of 25 mg., and was continued with 25 mg. intramuscularly every 4 hours. Within 24 hours, the body temperature (rectal) dropped from 38°C (101°F) to 31°C (82°F) and then down to 27°C (71·8°F) remaining at this point during the last 2 days of life.

On the 3rd day, diminished air-entry was noted on the left side, while a chest X-ray suggested a possible atelectasis at the left base. Despite the hypothermia and the state of unconsciousness, severe convulsions occurred on the 3rd evening. Curare was administered intravenously (7 mg.), resulting in complete muscular paralysis. Intermittent positive-pressure respiration (manual method) was carried out for about 6 hours.

The following day (4th day) the patient's condition deteriorated, with occasional convulsions and an increase of tracheo-bronchial secretion. Blood-electrolyte studies gave normal results.

On the 5th day disturbances in respiration made further curarization (7 mg. intravenously) and intermittent positive-pressure respiration necessary to maintain life. Urinary retention required catheterization.

An electro-encephalogram was attempted but, owing to technical reasons, it was not successful.

An electrocardiogram done 18 hours before death (body temperature 28°C) showed normal sinus rhythm with a rate of 80 per minute. A prolongation of the Q-T interval to 0·48 seconds was present in all leads. The pattern suggested hypocalcaemia (?).

The patient's condition gradually deteriorated, with poor air entry over the left chest, and death occurred on the morning of the 6th day in hospital.

Post-mortem examination revealed emphysema with patchy atelectasis of the right lung, and an almost complete collapse of the left lung. Thick purulent secretions were present in the trachea and bronchi, blocking the left main bronchus.

COMMENT

Any analysis of the result in a case of tetanus requires a careful assessment of the prognosis, since the severity of the condition may vary to a great degree from patient to patient. The factors to be considered are:

1. *Incubation Period.* If this is less than 7 days, the prognosis is bad; if more than 14 days, the prognosis is usually good. With an incubation period of 10 days, as in this case, the prognosis is equivocal.

2. *Period of Onset* (i.e. between first symptom and first convulsion). This is considered a more accurate index of severity than the incubation period.[1, 7] A period of less than 48 hours (in this patient the period of onset was 20 hours) carries a bad prognosis.

3. *Clinical Severity*. With severe and frequent spasms, involving the laryngeal and respiratory muscles, prognosis is poor. In this patient, the severity of the reflex spasms, causing cyanosis, and the temporary cessation of breathing, made radical treatment a necessity.

TREATMENT

Treatment was instituted along the following lines:

1. Rest, dark-room nursing, sedatives (chloral hydrate, 30 c.c. per day).
2. Excision of the septic wound.
3. Anti-tetanus antitoxin, a total of 300,000 units, of which one-half was given intravenously.
4. Antibiotics (penicillin and streptomycin) to prevent secondary infection).
5. Feeding by intravenous drip and then by an indwelling gastric tube.
6. Tracheotomy, carried out within 24 hours of admission, because of laryngospasm, difficulty in swallowing, and comatose condition of patient.
7. *Chlorpromazine* (Largactil) is considered to have a specific action on the neuromuscular endings.[5, 6] In addition, there is a sedative central effect occuring at a different level from that of the narcotics.[1] The atropine-like action helps in diminishing bronchial secretions, which is important in tracheotomized cases. Finally the hypothermia, probably due to peripheral vasodilatation, is considered to aid in treatment by diminishing metabolic needs.

Kelly and Laurence[6] used chlorpromazine alone in the successful treatment of a 2½-year-old boy, with severe spasms, an incubation period of 11 days, and a period of onset of 3 days. Over 16 days a total of 3,145 mg. was given (intravenously), as much as 330 mg. being given over one 24-hour period. Cyanotic episodes occurred, but tracheotomy was not performed. No mention is made of hypothermia resulting with these doses of chlorpromazine.

In our patient, aged 4½, a daily dose of 150 mg. of chlorpromazine combined with 30 c.c. of chloral hydrate resulted in a state of hypothermia and stupor which was maintained for 3 days, until death. It is doubtful whether the hypothermia was caused solely by the chlorpromazine, or whether other factors, such as central involvement, were concerned. In view of the rapid development of the state of hypothermia, we are inclined to consider that perhaps other factors (e.g. toxic or anoxic damage to the brain) contributed to the hypothermia.

8. Complete relaxation was not achieved with this therapy alone; on 2 occasions frequent spasms called for curarization with intermittent positive-pressure respiration, carried out manually for periods of 6-8 hours. Despite this therapy, our patient died of a massive atelectasis. We are, however, convinced that without this active form of treatment respiratory complications would not have allowed this patient to survive 5 days.

Was it possible to save this patient? In the light of further experience with respiratory problems in poliomyelitis, we believe the answer is probably in the affirmative. Success in critical cases of tetanus depends on the management of this difficult method of treatment.

SUMMARY

A fatal case of severe tetanus in a 4½-year-old boy, treated with tracheotomy, hypothermia, curarization, and intermittent positive-pressure respiration, is described. Death on the 6th day was due to pulmonary atelectasis. Despite the difficulty in treatment, it is considered that the way to better results lies along the methods attempted.

We are indebted to Dr. D. Benveniste, anaesthetist at Tel-Hashomer Hospital, for his invaluable assistance.

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EDITORIAL

BLOOD PRESSURE READINGS

What prognostic significance can be attached to raised blood pressure readings? And why is a raised blood pressure serious in one patient and not in another? These questions cannot be answered in the present state of our knowledge, yet they are of such everyday importance that every medical practitioner must hold some views upon them.

Measurement of the blood pressure is the pivotal point of the medical examination—no less to the average practitioner than to the examiner of service recruits, immigrants or university students. In this age of popular medicine, the examinee expects it; the doctor—be he an uncertain young fledgling or a tired specialist physician—often finds refuge in it when he does not know what else to do, for it is a concrete test that impresses the patient and therefore serves to gain his confidence. The value and importance of the test is magnified—perhaps *ad absurdum*—by the insurance companies, who employ doctors virtually to measure the blood pressure of their applicants and thereafter to check that there is nothing else wrong with them.

Anyone who has examined healthy young individuals will know how often the readings are above the traditionally normal levels of 140 mm. Hg systolic pressure and 90 diastolic. In a recent investigation carried out by a London cardiologist, Dr. William Evans,[1] 182 out of 400 army recruits (45%) were found to have systolic readings above 160, and 258 (64%) diastolic readings of 90 or over. The hearts of all these men were investigated by careful clinical examination, screening and electrocardiography, and pronounced normal, and they were accepted into the British army. About 10 years later 50 were re-examined, and in only 8 (16%) did the readings 'exceed slightly' the initial pressures. Both sets of examinations were conducted under as nearly ideal conditions as could be achieved: a quiet room, an unrushed consultation, the lowest of 3 readings being accepted as the basal value. There is nothing new in this type of work. Most medical officers engaged in it will bear out Dr. Evans' experience. Yet it would be a rash doctor who diagnosed essential hypertension in an able-bodied young man of 21 at a single examination. As Allbright emphasized more than 50 years ago, when he showed that hypertension could occur in the absence of renal disease, essential hypertension can be compatible with long life and freedom from serious disease.[2] On the other hand, no

VAN DIE REDAKSIE

BLOEDDRUK-LESINGS

Van watter belang is verhoogde bloeddruk-lesings by die prognose? En hoekom is 'n verhoogde bloeddruk ernstig by die een pasiënt en onbelangrik by die ander? Met ons hedendaagse kennis kan ons nog nie hierdie vrae beantwoord nie, maar die saak is van alledaagse belang, sodat elke geneesheer wel die een of die ander mening daarop nahou.

Die meet van die bloeddruk is die spil waarom 'n mediese ondersoek draai—ewe belangrik vir die gemiddelde praktisyn as vir die ondersoeker van diensrekrute, immigrante of universiteitstudente. Die pasiënt verwag dit in hierdie eeu van leke-medisyne; die geneesheer, of hy nou 'n onsekere jong doktertjie of 'n moeë spesialis-internis is, neem dikwels sy toevlug daartoe as hy nie weet wat anders om te doen nie, want dit is 'n tasbare toets wat 'n indruk maak op die pasiënt en dus sy vertroue wen. Die waarde en belangrikheid van hierdie toets is baie vergroot—miskien *ad absurdum*—deur versekeringsmaatskappye, wat feitlik dokters huur om die bloeddruk van hul applikante te meet en om daarná na te gaan of hul iets anders ook makeer.

Enige dokter wat reeds gesonde jong mense ondersoek het weet dat die lesings dikwels bó die normale hoogtes van 140 mm. Hg sistoliese druk en 90 diastolies is. In 'n onlangse ondersoek deur 'n Londense kardioloog, dr. William Evans,[1] was dit bevind dat 182 uit 400 (45 persent) rekrute vir die leër sistoliese lesings bo 160 gehad het, en 258 (64 persent) diastoliese lesings van 90 of meer. Al hierdie mans het noukeurige kliniese hartondersoeke ondergaan, met fluoroskopie en elektrokardiografie, en dit is bevind dat hulle normaal was. Hulle is in die Britse leër opgeneem. Omtrent 10 jaar later is 50 van hulle weer ondersoek, en by slegs 8 (16 persent) het die lesings die oorspronklike syfers ‚effens oorskry'. Beide ondersoeke is onder feitlik ideale toestande uitgevoer: 'n rustige kamer en 'n ongejaagde mediese ondersoek; en die laagste uit 3 lesings is aanvaar as die basale waarde. Daar is niks nuuts in hierdie soort werk nie. Die meeste mediese beamptes wat daarby betrokke is, sal dr. Evans se ondervinding kan bevestig. Maar die dokter wat tydens 'n enkele ondersoek essensiële hoë bloeddruk by 'n gesonde jong man van 21 jaar oud diagnoseer, is heeltemal te haastig. Soos Allbright meer as 50 jaar gelede benadruk het, toe hy bewys het dat hoë bloeddruk *sonder* niersiekte kan voorkom, kan essensiële hoë bloeddruk gepaard gaan met 'n lang lewe sonder ernstige siekte.[2] Aan die ander kant hoef geen dokter daaraan herinner te word nie dat egte slagaarontaarding, met sy onvermydelike nasleep van ernstige komplikasies, een van

doctor need be reminded that true arterial degeneration, with its inevitable train of grave complications, ranks high in the list of causes of human mortality and morbidity. Organic hypertension is due to arterial degeneration—the relatively malignant type with arteriolar necrosis, the relatively benign type with atheromatous changes—and the problem is this: How does one know whether a patient with a clinically demonstrable high blood pressure is going to develop irreversible arterial changes that will shorten his life, or whether he is of the type of human being that normally shows a rasied blood pressure in the consulting room and will live to a ripe age?

Evans seeks to separate the condition of 'labile and intermittent hypertension which may manifest itself under the stress of medical examination' (which he calls 'hypotonia') from the text-book disease of hypertension. While most people will commend his notion of 'hypotonia', Evans will find many critics of his assertion that it can actually be distinguished in advance from true hypertension. As his own series showed, cardiac pain was not confined to the true hypertensives (nearly 50% of his healthy group complained of it 10 years later) and some of his cardiac 'hypotonics' died of coronary infarction. Moreover, American work has shown that sustained hypertension is commoner in persons with previous transient hypertension than in those whose blood pressure has never been raised.[3] One wonders whether Evans' follow-up period of 10 years is sufficient; perhaps the passage of a further decade will alter the picture entirely and render the fate of his 'hypotonic' group nearer to that of his hypertensives.

It is clear that there is as yet no solution to this problem. Experience and common-sense remain, in the absence of other signs and symptoms, the best guides to when a raised blood-pressure reading should be regarded as significant. The best place for your sphygmomanometer when examining healthy young adults—according to one prominent physician—is the back seat of your motor car, but sooner or later a coarctation of the aorta, a hydronephrosis, or some other remediable condition will be missed. And when dealing with patients the blood pressure must undoubtedly be taken—for 3 reasons, viz. the patient expects it, the examination may help to establish a confidence and, finally, it may be useful in the diagnosis. It is particularly useful in reassurance, i.e., as a means of enabling one to tell the patient that his pressure is 'normal'; with the present-day lay ideas of the meaning of 'low blood pressure' and 'high blood pressure' it is probably unwise ever to tell him anything else. Certainly lesser mortals than Dr. Evans will have difficulty in persuading any selection board, let alone an insurance company, to accept a young man with a blood pressure of more than 140/90 as a perfectly fit individual.

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die mees voorkomende oorsake van menslike sterftes en siekte is. Organiese hoë bloeddruk word veroorsaak deur slagaarontaarding—die betreklik kwaadaardige soort met endslagaar-ontaarding, en die betreklik goedaardige soort met slagaarvervetting—en die probleem is dit: Hoe weet 'n mens of 'n pasiënt met 'n klinies bewysbare hoë bloeddruk later onherstelbare slagaarveranderings gaan ontwikkel wat sy lewensduur sal verkort, en of hy van die soort is wat gewoonlik 'n verhoogde bloeddruk in die spreekkamer toon en tog 'n lang lewe sal geniet?

Evans trag om die kondisie van ‚onvaste en onderbroke hipertensie wat onder die stremming van 'n mediese ondersoek na vore kom' (hy noem dit ‚hipotonie') te onderskei van die hoë bloeddruk wat in die handboeke beskryf word. Die meeste mense sal wel Evans se teorie van ‚hipotonie' goedkeur, maar daar sal baie wees wat krities sal staan teenoor sy bewering dat dit werklik vooraf kan onderskei word van die egte hoë bloeddruk. Sy eie reeks toetse het bewys dat pyn in die hart nie beperk was tot lyers aan egter hipertensie nie (byna 50 persent van sy gesonde groep het 10 jaar later van hartpyn gekla) en sommige van sy ‚hipotoniese harte' het aan hart-infarksie gesterf. Ook is dit in Amerika bewys dat volhoue hipertensie meer dikwels voorkom by pasiënte wat tevore verbygaande hoë bloeddruk getoon het as by dié wat nooit hoë bloeddruk gehad het nie.[3] 'n Mens wonder of Evans se opvolgperiode van 10 jaar lank genoeg was; miskien sou die beeld ná nog 'n dekade heeltemal anders lyk, en sal die lot van sy ‚hipotoniese' pasiënte meer op dié van sy lyers aan hoë bloeddruk lyk.

Dit is duidelik dat hierdie probleem nog nie opgelos is nie. Wanneer ander tekens en simptome uitbly, moet ons maar op ons ondervinding en gesonde verstand staat maak om te bepaal wanneer 'n verhoogde bloeddruklesing as veelbeduidend beskou moet word. Die beste plek vir die bloeddrukmeter is agter in jou motor, maar vroeër of later sal 'n saamgeperste aorta, 'n geval van hidronefrose, of die een of ander geneesbare siekte oor die hoof gesien word. En wanneer die dokter met pasiënte te doen het, moet die bloeddruk altyd gemeet word—om 3 redes: die pasiënt verwag dit, die ondersoek dra moontlik by om sy vertroue in die dokter op te wek, en laastens kan dit behulpsaam wees by die diagnose. Dit is veral nuttig aangesien dit die dokter in staat stel om die pasiënt gerus te stel dat sy bloeddruk ‚normaal' is. Maar met die hedendaagse leke-opvatting van ‚lae en hoë bloeddruk', is dit miskien raadsaam om maar altyd aan die pasiënt te sê dat sy bloeddruk normaal is. Dit staan vas dat minder belangrike mense as dr. Evans dit moeilik sal vind om enige keurraad—om nie eers van versekeringsmaatskappye te praat nie—oor te haal dat 'n jong man met 'n bloeddruk van meer as 140/90 heeltemal gesond is.

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HEPATIC FAILURE

An important function of the liver is the detoxication of products of intestinal protein digestion that reach it through the portal vein. The break-down of this mechanism is believed to be the cause of the characteristic syndrome of hepatic coma.[1] Entry of undesirable protein metabolites into the systemic circulation may take place in two ways; firstly as a result of massive hepatocellular destruction (as, for example, in fulminating viral hepatitis) these substances are

allowed to traverse the liver unmodified by normal metabolism, and secondly through the presence of an extensive collateral circulation which enables them to by-pass the liver.

The exact nature of the harmful products remains to be settled. Ammonia has been blamed, but blood-levels correlate poorly with the clinical state.[1] However, neurological changes are always present when the level exceeds twice the normal upper limit of 1 μg./ml.[4] The technique of estimation is too cumbersome for routine use.

Prominent early manifestations of this 'portal-systemic encephalopathy' are personality changes and constructional apraxia. The patient often has difficulty in reproducing simple designs in matches, and the handwriting deteriorates and becomes irregular (a daily handwriting chart is useful in following slight fluctuations in the status of the convalescent patient). Non-specific electro-encephalographic changes are seen. When present, associated signs, e.g., flapping tremor, foetor hepaticus, spider naevi and palmar erythema, not to mention jaundice, help in diagnosing coma or the earlier neurological changes as being due to liver failure.

In cirrhosis the patient is often in a precarious state of balance, easily upset by an increase in the nitrogenous content of the blood. This is usually due to a large protein load in the bowel, e.g., a high-protein meal or a gastro-intestinal bleed. Methionine is known to precipitate coma in the susceptible, and ammonium chloride, given as a diuretic, is dangerous in these people. Acetazolamide (Diamox) may also induce coma, but does so by inhibiting tissue uptake of ammonia. The dangers of operations and acute infections in cirrhotics are also well recognized.

In the emergency treatment protein should be banished from the diet, and enemas and saline purges used to flush nitrogenous material from the gut. An attempt should be made to inhibit bacterial digestion of protein by bowel sterilization with oral chlortetracycline or neomycin.[2] If gastro-oesophageal varices continue to bleed, tamponade with a Sengstaken balloon should be attempted.

At least 1,600 calories should be provided daily, as glucose; if the oral route is impractical, a 20% solution can be infused into the superior vena cava through a polythene catheter. When the patient recovers protein is slowly reintroduced into the diet, but a cirrhotic who has had a bout of coma should not take more than 50 g. per day. The well-compensated cirrhotic with ascites who has not had coma falls into a different category; a high-protein diet plays an important part of the management in this type of case.

It has been stated that various 'specific' substances alter the course of hepatic coma. Glutamic acid combines with ammonia to form glutamine; arginine,[3] ornithine and thioctic acid are supposed to stimulate the tricarboxylic acid cycle. Their advantages are mainly theoretical and there is not enough evidence to recommend any of them for routine use. Sherlock[4] considers that there is no place for steroids in the treatment of hepatic coma due to cirrhosis, where the principles outlined above usually achieve their purpose; fulminating hepatitis has such a high mortality that they should be tried, in addition to the standard regime, if only for want of something better to do.

1. Annotation (1957) : Lancet, **1**, 623.
2. Annotation (1957) : *Ibid.*, **2**, 280.
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THE PARASITIC DISEASES OF MAN IN AFRICA *

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In opening the discussion on parasitic diseases of man in Africa I intend to adopt a broad definition of what are parasitic diseases by including not only those caused by protozoan and helminthic parasites but also other diseases in which the mechanism of transmission to man involves an intermediate host.

Broadly speaking the clinician practising in tropical Africa may divide parasitic diseases into 2 groups, the first composed of those which are more or less widely distributed and the second those with a focal distribution.

Diseases with a Widespread Distribution

Malaria, bilharziasis and hookworm fall into the first category for, although there may be regions in which they are absent, they must for practical purposes, be considered by every doctor engaged in clinical work in any part of tropical Africa. The frequency and distribution of malaria depends largely on the climate, acting through the mosquito vector; directly and particularly on rainfall and temperature and indirectly on altitude, since increase in altitude brings about a decrease in temperature and a more efficient natural drainage through swiftly flowing rivers. Other subsidiary influences also to be taken into consideration in certain areas are: (*a*) the distance from the sea or other large expanses of water, (*b*) ocean currents, (*c*) the direction of prevailing wind, and (*d*) the existence of forests.

In an equatorial climate the transmission period for malaria can be expected to last throughout the year, but this again will vary with the rainfall and altitude. As the altitude increases the transmission period becomes shorter. At 4,500 feet the incidence of endemic malaria is still heavy, but around 5,600 feet the epidemic type becomes the rule. It is even described up to 8,600 feet. Although malaria may be more frequent in low-lying areas it does not follow that it is more severe than the type encountered on the plateaux from 2,000 to 5,000 feet above sea level, where severe sporadic cases of virulent outbreaks are known to occur. The reason for this is that at the coast the population is exposed to the infection more or less throughout the year. This enables the individual to maintain a tolerance to a greater or lesser

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degree. But on the plateaux the transmission period is much shorter and after a long dry season much of the immunity built up in the previous rainy season has been lost by the time the following rains arrive.

There are few rivers in the centre of Africa free from bilharzial infection and the greater the rainfall the more the disease is encountered. In Mashonaland for example there is far more bilharziasis than in the drier and more arid parts of Matabeleland. Schwetz (1951) found *S. mansoni* at an altitude of 6,300 feet, but in Southern Rhodesia it is said not to occur above 6,000 feet. Inyanga, for instance, is believed to be free of the disease. Hookworm disease, too, is more frequent in lower areas than in higher regions where the water drains off more rapidly. But other factors come into play at the same time. A high temperature is more favourable to the parasite, which is also found more often in a clay soil than in the sand veld.

Parasites with a Focal Distribution

In contrast to those with a wide distribution, we meet parasites that are confined to focal areas, and the factors which determine this are largely unknown. Sleeping sickness, plague, kala-azar, yellow fever, amoebic disease and filariasis are examples of this. In some places a particular disease is common, yet in others it is rare. For instance the pathogenic amoeba is very common in and around Durban, where the peri-urban Africans seem to suffer more than the Europeans or Indians. It is possible that malnutrition may be a factor, or perhaps an increased virulence of the parasite, because not far away along the coast lies Lourenco Marques, where the disease is not encountered with the same frequency or intensity. Further, even in Southern Rhodesia I have noticed its incidence seems to vary almost from town to town. It is possible, of course, that the apparent difference may result from the varying skill of the technician. Judging by the frequency of liver abscess in my African patients I am inclined to believe that amoebic disease is commoner than many people believe.

Plague too is puzzling. It is seen fairly regularly in Barotseland and in some parts of Bechuanaland, but in Southern Rhodesia, which is adjacent to both these territories, no case has been notified since European settlement of the country, although the appropriate rodent reservoir exists there.

Although ascariasis, the common parasite of the human intestine, is encountered in most parts of Africa, it is particularly prevalent in some regions. In a small survey in Ibadan Jelliffe (1951) found that over 80% of house servants harboured this parasite. He recorded cases of pulmonary eosinophilia, intestinal obstruction, perforation of the bowel, and obstruction of the common bile duct and trachea, due to this worm. In Mashonaland cases are not nearly so common and the complications are only rarely encountered, but in Manicaland the frequency is very much greater.

We know a little about the effects of an alteration in the environment of a parasite. Not all parasites are affected in the same way, but interference in the natural surroundings of some definitely influences their life cycle and thus their frequency in man. The organization of a civilized community in a malarial area tends to drive out the anopheline mosquito. Similarly, the clearance of vegetation in a trypanosomiasis area often disturbs the tsetse fly. This was evident in the Hartley district of Southern Rhodesia in the first decade of this century, when the cutting down of trees for the Cam and Motor Mine near Gatooma drove back the fly. The clearing of banks of rivers in Gambian sleeping-sickness regions removes the habitat of the tsetse fly. It is believed by some that game elimination leads also to the eradication of the fly.

But with bilharziasis interference with nature produces the opposite effect. The snails multiply with the digging of new dams, and the establishment of irrigation projects spreads the infection yet further afield.

Over the years Bancroftian filariasis seems to have existed continuously in certain defined pockets. When David Livingstone sailed down the Zambesi in 1856, he recorded the frequency with which the Natives suffered from elephantiasis of the limbs and scrotum. Today we see many cases from the same region complaining of the same complications, including orchitis, epididimo-orchitis, hydrocele, funiculitis, lymph scrotum, and even chyluria. The disease does not seem to have spread beyond these confines to Mashonaland where the same mosquito vectors are known to exist.

Sites of Election

Besides their focal distribution, many parasites, as Heisch of Kenya (1956) has stressed, select special local sites in which the disease may be contracted. For instance, relapsing fever results from the ticks that inhabit Native huts and plague is spread by the domestic rats which pillage the grain bins in the kraal after they themselves have been infected by the wild rodents which live in burrows in the fields. It is only when the wild rats are numerous that *P. pestis* reaches *Rattus rattus* and so man. In Kenya the sites infected with kala-azar are those round termite hills from which emerge the sand-flies responsible for this disease.

Heisch points out that the sites of infection may be relatively stable or unstable. Unstable areas he calls ecotones. Transition zones like forest edges and clearings and cultivated plains, are excellent examples of ecotones, which imply states of tension in nature, in which the ecological balance is liable to be upset. The balance is disturbed in a plague-infested area when the fields are ploughed, and the disease tends to disappear. Transition zones are important meeting-places for man, animals and insects, and result in an interchange of parasites. An excellent example of this is shown by workers in Uganda, where the mosquito *Aedes simpsoni*, the vector of yellow fever, lives in the forest edges. In the forest dwell the tree-top mosquitoes bearing the virus with which they infect the monkeys. The monkeys appear at the edges of the forest to raid the banana plantations and infect the *Aedes simpsoni*, which in their turn affect man, who comes into contact with them.

On the other hand some sites of infection known as 'habitat niches' are stable. Instances of these are Native huts, termite hills and even animal burrows that are away from interference by man. Even forest edges and other transition zones may be relatively stable.

Disappearance and Reappearance of Parasitic Diseases

Again, for reasons not yet clear, a parasitic disease may disappear from a region or become very rare, or it may appear in increased intensity in some other area far removed from its customary focus of infection. For instance, a very severe outbreak of yellow fever occurred in the Nuba mountains

in the Sudan, in which hundreds of Africans lost their lives. After the last war, too, a significant outbreak of kala-azar was reported in the Kitui district and another at Marigat, in the Rift Valley in Kenya, where hitherto this disease was rarely seen, in contrast to the Sudan, which is the accepted focus for kala-azar. After some investigation it was suggested by Heisch that sandflies emerging from termite hills were responsible.

Equally interesting is the tremendous diminution in the incidence of tropical ulcer and tropical myositis, which have largely disappeared excepting in small isolated foci. In the days of Livingstone and for some years later these two diseases were amongst the most serious tropical conditions with which one was called upon to deal. Although they have been linked with a poor diet, the possibility of a parasitic origin has not been disproved.

CLINICAL ASPECT

Lack of Specific Pathological Changes

In countries where parasitic disease is not of major importance the clinician is aided in his diagnosis in a large number of day-to-day diseases by a confident pathological report. Many of the non-parasitic diseases produce specific histological changes. With the majority of the parasites, however, confirmation of the diagnosis of the cause of an illness and often even of a death may be impossible. It is difficult at autopsy to attribute the cause of death to relapsing fever or hookworm disease.

Most authorities do not agree with the high number of deaths attributed at autopsy to malaria in infants and young children in hyperendemic regions, since most infants at this time of life harbour the parasite and it may be coincidental with the presence of broncho-pneumonia, anaemia or other disease. Unless plugging of the capillaries of the brain with malarial parasites can be demonstrated, there is no definite proof that malaria is the cause of death in an indigenous population in many of whom the parasite is habitually harboured and in whose tissues the pigment is present. The same may be said of sleeping sickness. Filariasis, too, produces indefinite changes.

Bilharziasis may be regarded as an exception in this respect, since pathological changes with eventual fibrosis occur at the site of ovideposition. But even here the problem is by no means as simple as it might appear, because of the widespread deposition of ova in the different organs of the body. Many diseases occur coincidentally with bilharziasis in an organ, and as a result it is difficult to be certain whether or not the bilharziasis produced the lesion. Fibrosis in the ureter can be accepted as due to the ova because few other diseases are likely to affect this part of the body. But the finding of ova in a multilobular cirrhotic liver is a different matter, because a large section of the population has ova in the liver without cirrhosis. Many authorities, especially in Egypt and South America, are convinced that cirrhosis of the liver commonly follows bilharzial infiltration. They may be correct, but it is only fair to stress that cirrhosis of the liver occurs in parts of Africa where bilharziasis does not exist, and furthermore appears with the same intensity. The same may perhaps apply to cardio-pulmonary schistosomiasis, although here I am on less certain grounds. It is said that in Egypt some 2% of infected bilharzial subjects die of right heart failure due to arteriolar obstruction by ova deposited in them as emboli. The pressure in the pulmonary circulation is raised and varying degrees of heart strain commonly follow. Just as in the liver, over 50% of bilharzial subjects have ova in their lungs with apparently no ill-effects. We know that right heart failure from pulmonary hypertension of diverse causes occurs in non-bilharzial regions and there is little reason to suppose that these cases are not seen in bilharzial territories. It may be argued, therefore, that the right heart strain is incidental to the ova present in the lungs. A point favouring a bilharzial arteriolitis, however, is the angiomatoid formations which are said to be almost pathognomonic of bilharziasis.

Clinical Picture in the European and Bantu Patient

It is difficult to give an adequate description of a tropical disease which is generally applicable, because the disease usually manifests itself differently in the European and the Bantu. On the whole, the European is virgin soil for infection. When he becomes ill with a parasitic infection this generally occurs in a body which has never had the infection before and is free from other tropical diseases. He is a non-immune subject or one who has not been sensitized by previous infections. A truer picture of the acute uncomplicated effects of the disease can be obtained from him than from the Bantu, who has been exposed to parasitic infection from childhood and has acquired a hereditary racial or personal resistance to the disease.

It is not easy to draw a satisfying clinical picture of malaria, sleeping sickness, yellow fever or bilharziasis in the Bantu. The body responds differently according to its state of immunity to the particular parasite. No two individuals are alike. When an African (and perhaps his parents too) has been brought up in an area in which he has not been exposed to a particular infection, he is liable on contracting that infection to suffer from it in the same way as a European and thus for instance may as easily die of cerebral malaria and blackwater fever.

The clinical picture varies from year to year, season to season and district to district, and what makes it still more difficult to assess, in the African, is that he harbours more than one parasite at a time, sometimes even 3 or 4. Thus in endemic regions with a multiplicity of infections how can one adequately unravel the symptoms resulting from each? This is probably the reason why no clear cause has yet been established for the common tropical anaemia of Africa, despite the many interesting publications recently issued. Malaria, bilharziasis and hookworm disease all affect the haemopoietic system and, to complicate matters still further, the indigenous population exists on a diet which may be lacking in one or more factors essential in the manufacture of the red cell.

The Diagnosis

In England one might say that a clinician can practice satisfactorily with his stethoscope. In Africa he needs the microscope as well. He cannot move without it, else he misses the many parasites. Fortunately with the use of a microscope we can recognize the great majority of infections, such as malaria, bilharziasis, relapsing fever, sleeping sickness and filariasis, especially in the more acute stages of the illness. But the difficulty in the African is to know whether the presence of the parasite accounts for the symptoms. We know that malarial parasites are present in both febrile

and afebrile patients. The parasites may be extremely numerous in a child and yet he may be running round quite unconcerned. It is difficult to lay down a rule in the diagnosis of parasitic disease but, on the whole, it may be said with reasonable safety that, in an African, if a parasite is demonstrated and no other cause can be found for the symptoms the diagnosis of that parasitic disease may be made.

Bilharziasis is interesting in that it is one of the very few parasitic diseases in which stress is laid on the finding of viable eggs in order to establish an active infestation. When the ova are dead it is assumed that they are discharged in the same way as a foreign body and the individual is cured. Yet in other diseases no one insists on proving whether the organisms are alive or dead when found in the discharges. I personally can see very little reason for supporting such a point of view and maintaining that the bilharzia ova should be alive. There are two good reasons against this. Firstly, in urinary bilharziasis large numbers of terminal-spined ova are deposited in the rectal mucosa and yet it is exceptional for them to be discharged in the stools. Secondly, in many cases ova are deposited in the lungs in close vicinity to the alveoli, but are rarely found in the sputum. If these were foreign bodies, why are the terminal-spined ova not more commonly found in the stool or the ova of either variety in the sputum? It would seem to me that the ova of *S. haematobium* appear in the urine because it is part of the life cycle. The finding of dead ova does not imply in my opinion, that the worms are dead, but that the ova died soon after discharge.

TOLERANCE IN THE AFRICAN

Bilharziasis

It is convenient at this stage to discuss briefly the tolerance of the indigenous population to some of the main parasitic diseases. Our knowledge of this subject is by no means complete, but of special interest is the extraordinary battle put up by the host against most of the tropical parasites. The host does not appear ever quite to overcome the infection, except at certain periods of his life. The immunity is not as clear cut or as firm as in many non-tropical diseases, such as measles, poliomyelitis or typhoid fever. Many believe that the immunity, at least in malaria, is related to the nutritional status of the individual. At times, particularly with lack of protein, the amount of available globulin may be reduced and the infection may reappear in a severer form. Thus in hyper-endemic regions the lower protein intake is given as a reason for the increased frequency of malaria in the later months of pregnancy. With most of these tropical parasites we are not sure what happens with re-infection. Does a person with active bilharziasis become re-infected with more worms every time he exposes himself to infected waters or is there a limit to the amount of worms the body will accept? Does a state of immunity develop later and does the infant acquire some immunity from its mother? In my experience, it appears that in bilharziasis a person acquires a heavier infection with repeated exposures. How else can we explain the grosser lesions found in the Bantu as compared with the European, in whom the infection is usually light (Honey and Gelfand, 1957)? For example marked fibrotic thickening of the bladder and ureter, leading to hydronephrosis, is a very frequent occurrence in the Bantu. Compared with the African, the European exposes himself only occasionally. Experimentally, Vogel (1945) demonstrated that in monkeys (*Macacus rhesus*), after repeated exposure to the cercariae of *S. japonicum* over many months, there was a progressive decrease in ova. An immunity to superinfection was finally established so that a dose of cercariae which previously would have been fatal proved quite innocuous. Standen (1949) also observed a similar immunity in the *Macacus rhesus* monkey exposed to the cercariae of *S. mansoni*. Recent experiments by Lurie and De Meillon (1957) with mice infected with repeated small infections show that, once the mouse is infected, subsequent infestations do not aggravate the existing state. The initial dose of cercariae has an immunizing effect against further doses. Fairley (1951) states that there is no evidence that immunity of this type results in man from repeated exposure to schistosome cercariae over a number of years. He rightly draws attention to the importance of this subject, for if an immunity to super-infection exists in man, it might be preferable to employ a drug producing clinical rather than radical cure in heavily infected areas, particularly if it could be taken orally. In this way, I think, Miracil has a distinct future in *S. haematobium* infections. With this drug the load of worms would be reduced possibly to a sub-clinical level so that the body with a smaller number would be able to resist further infection.

In endemic areas the highest incidence of bilharziasis is in children from the ages of 4 to 15, the number passing ova becoming less in adulthood. In Southern Rhodesia Dr. Morley Smith and I investigated its incidence in infants. Out of 27 infants tested, we found only 2 showed positive results, whereas in the 63 children we examined between the ages of 4 and 12, 37 passed ova. Workers in other territories have also found a low incidence in infants and a high one in children. At first we wondered if the infant was not exposed to infection in the same way as the child. We reasoned that the mother might be afraid that it might be drowned or injured by crocodiles. But on closer investigation, we found the mother bathed her infant in pools, especially when she was doing her laundry. It may be argued, therefore, that the infant has some immunity or protection against contracting the disease. We carried out a similar investigation at the other extreme of life and found that few elderly adults in the same region passed ova, despite their exposure to infection. It is probable that, by this time of life, they have acquired an immunity by which the body is able to reject invasion of the cercariae. Thus there appears to me to be some evidence that an immunity can be established in the body through infection and possibly a temporary one through the mother to her infant. An argument against this is that in other diseases, such as diphtheria, the immunity of a babe rarely lasts longer than 6 months or a year or two.

Hookworm Disease and Filariasis

I am not sure what the immune processes are in hookworm disease. As in bilharziasis, it is probable that there is a limit to the number of worms that can infest the body. As far as I have seen it at autopsy in Rhodesia, the infestation has been light, unlike Uganda, where hundreds of worms may be found in any one individual. In Gambia, MacGregor and Dean Smith (1952) have shown that the incidence of hookworm disease is very low in children up to the age of 5. They argue that the young child is not permitted to wander far from its hut and is left behind when the mother goes into the bush on her calls to nature. So far my experience con-

firms that of the workers in Gambia, but I would suggest that again the reason may be some protective power against the infection in the infant and young child.

Filariasis, especially that due to *Wuchereria bancrofti* and *Onchocerca volvulus*, is rare before the age of 4 in endemic regions. Dean Smith and MacGregor found that the first manifestations of bancroftian filariasis did not appear before the age of 7. They suggest that one of the reasons for this may be that, as malaria is usually present in the same region, the young child suffers from a continuous fever which protects it from acquiring filariasis. Is it possible to postulate a similar type of immunity for bilharziasis, filariasis and hookworm disease?

Malaria

Malaria is different in that it is the infant who seems to bear the brunt of the infection in a hyper-endemic region. Afterwards the child is able to survive the attacks and by the time he reaches 15 years is probably so resistant that, unless the infestation is extremely heavy or virulent or there is some other good reason, he is not likely to suffer much sickness or risk of death from the disease. The infant up to the age of 3 months rarely shows parasites in the blood, but by the age of 1 or 2 years most infants have had the disease. The immunity conferred on the infant by its mother stands it in good stead, not only for the first 3 months, but even later, for the attacks tend to be milder in all respects than those suffered by European children without that immunity. There is much controversy about the number of infants who die from acute malaria. The work of Garnham in East Africa in recent years shows that, in actual fact, fewer die from the disease than was formerly believed. Yet Bruce-Chwatt considers that 5% of children under 5 die directly from fever each year. Another school of thought believes that, whilst there may not be many direct deaths from malaria, the disease is an aggravating factor in many of the deaths in infants and young children from gastro-enteritis, broncho-pneumonia, tuberculosis, anaemia and even kwashiorkor, by lowering of resistance. In my experience, malaria rarely kills the African infant, and I am doubtful whether it is an important factor in the morbidity rate. The immunity of the baby in its first few months of life must be very high in hyper-endemic regions, since congenital malaria is rare. But other factors may be responsible. There is experimental evidence that breast milk renders an attack of malaria less severe and it is possible that in certain regions the possession of the sickle-cell trait may protect the infant against the disease.

It has been suggested that a raised body temperature in a hyper-endemic zone is an indication of malarial infection, but on closer investigation, Colbourne (1955) showed that this elevated temperature in children up to the age of 14 years is not due to malaria, but is a normal occurrence. He is careful to point out that we do not know what is the normal temperature in the Bantu. He treated febrile school children of the age of 7 with antimalarial drugs, but they did not appear to gain any tangible benefit from the treatment except perhaps a slight increase in weight and less absenteeism.

The effect of malaria on the foetus has also been investigated by workers in Africa. At one time it was believed that abortion was likely to result when a pregnant woman contracted malaria. I think this is the case in a non-immune mother and possibly in an immune one when the infestation is heavy. But workers have found little or no increase in the abortion rate in hyper-endemic areas during the malaria season. The same findings apply to the stillbirth rate. But in a hyper-endemic zone the incidence of malaria in pregnant women towards the end of parturition is approximately double that of the general population. It may be that her resistance has been reduced by the strain of the pregnancy and she is then more liable to the infection than the ordinary adult.

An excellent example of acquired immunity to malaria by the African is shown by the frequency of *P. malariae* and *P. vivax* in infancy and early childhood, after which these two strains disappear and are rarely found in the adult (Schwetz, 1949). The African appears therefore to develop an immunity to these two parasites, but is not quite able to protect himself from the malignant tertian form of the disease.

In a hyper-endemic zone children, on the whole, suffer little disability from malaria. It is believed that people moving from an endemic area to a non-endemic one lose their immunity and on their return suffer severely when re-infected. But Colbourne (1955) investigated this subject by studying Gold Coast students who had spent some years in England and found they did not suffer unduly on their return. They contracted no severe attacks and managed to control the fever quite readily. We are also taught that Africans who move from one endemic region to another are liable to suffer acute attacks of malaria owing to their coming into contact with a different strain of parasite (Schwetz, 1949). While this may be so, I have not noticed it in Africans coming to Mashonaland from Northern Rhodesia, Nyasaland and Portuguese East Africa.

Garnham (1949) classifies immunity in malaria into 3 types :

1. *The racial type*, in which the individual has lived in the area for generations and built up an immunity. This is long lasting in contrast to Africans brought up in non-endemic areas, who suffer severely on moving to an endemic one.

2. *The transmitted or passive immunity* which is passed on from the mother to her child and lasts only about 3 months.

3. *An acquired immunity (premunition)* for which, for the purposes of this paper, I prefer to use the word tolerance.

Relapsing Fever and Sleeping Sickness

Relapsing fever bears some similarity to malaria in that the infant is frequently infected. Bell (1953) from East Africa reports that the incidence of this disease varies inversely with the age of the patient. In infants and children, too, the prognosis is more serious. Differing in this respect from malaria, congenital relapsing fever has been described a number of times in endemic regions.

Even in both the Rhodesian and Gambian forms of sleeping sickness, there seems to be an immunity to this disease, which is normally regarded by many as fatal. Mild cases are frequent in sleeping-sickness belts and carriers of the rhodesiense type of the disease have also been recognized. It is known that a mild case may sooner or later be complicated by involvement of the nervous system, a development which may occur at any time—even months or years after infection.

Summary

Thus to summarize, the clinical picture of the main parasitic disease of Africa depend on complicated immune

processes. The Bantu has acquired a racial form of tolerance to these diseases, producing a clinical picture quite different from that of the European living in these parts in whom the clinical features are more distinct and more uniform.

MALNUTRITION, ANAEMIA AND PARASITIC DISEASES

An important subject which should be discussed is the effect of these parasitic diseases on the nutritional state of the body. Many workers, including myself, have said that bilharziasis, malaria and hookworm disease reduce the appetite and induce debility and, in this way, tend to hasten the onset of nutritional disease in a population whose diet is already on the borderline or distinctly lacking in proteins and vitamins. Furthermore, these diseases often cause either a loss of blood or blood destruction and so are considered responsible for the anaemia so common in these people. Recent evidence, based on some interesting surveys, has given rise to doubt whether nutritional disease or anaemia is in fact hastened by these diseases. A useful research was that by MacNamara (1948), who carefully investigated a number of school-children of the same social grade in Central Nigeria, where bilharziasis, hookworm and malnutrition are frequent. One group of children was given a good diet, another was treated for parasitic diseases, and the third, the control group, was left to carry on in the normal way. After an interval of 100 days MacNamara found that the children on the good expensive diet had gained the most weight—an average of 1 lb. 10½ oz. Those treated for helminths had gained an average of 9½ oz. and the control group also very little. All signs of malnutrition had disappeared from the well-fed group, but there was no change in the nutritional state of those who were deparasitized. He found that the children on the good diet showed the best results in endurance tests, but their teachers noticed no difference in their mental ability. In this connection, in South Africa, Kieser (1934) maintained that the child with bilharziasis was lethargic and had poor powers of concentration, so that his school work suffered. In Southern Rhodesia work on similar lines was recently carried out by Blair *et al.* (1949), who found that the children with bilharziasis showed better school results than those free of the disease.

Beet (1949) investigated the haemoglobin levels of children in the Lala district near Broken Hill in Northern Rhodesia, where bilharzia, hookworm and malaria are endemic. He first compared the mean haemoglobin levels in children with hookworm and those without and found no difference (12·3 g.% in both groups). He therefore concluded that hookworm disease was not an important cause of anaemia in these people. He then compared a large group of children with *S. haematobium* with a similar group without the disease and found the average haemoglobin (11·6) of the children with bilharziasis was lower than that of those without (12·5). He considered, therefore, that bilharziasis tended to lead to slight anaemia. But Gerritsen *et al.* (1953) in Johannesburg did not find much anaemia in the 8 Africans with obvious haematuria in whom they studied the degree of blood loss and reached the conclusion that bilharziasis was not an important factor in the production of anaemia.

Colbourne, Edington and Hughes (1950) in West Africa approached the problem from a different angle. They compared the incidence of hookworm disease, bilharziasis, malaria and ascariasis in children with deficiency disorders and those without signs of malnutrition and found no difference. Thus from these surveys, it would appear that the importance of parasitic diseases in the causation of malnutrition has been over-stressed in the past.

So far we have tried to assess the harm that the parasite can cause man. We are agreed that each one can lead to disease, but we are not sure whether it influences the nutritional state of the body. Do the parasites in any way protect the body or confer any benefit to the individual? It could be argued that malaria, bilharziasis, sleeping sickness and kala-azar produce an excess of globulins in the blood, which may confer an immunity to bacterial infection. But this does not seem to be the case. Does the malarial fever so frequent in children in an endemic area protect them against nephritis or does a malarial infection in an adult African render him less liable to general paralysis of the insane? In my experience both these diseases are sufficiently common to lead me to believe that the fever does not confer this protection. Nor am I convinced that parasitic diseases protect the body against other infections such as pneumonia and tuberculosis.

A BALANCED APPROACH

There are a number of rival schools of thought, each stressing the responsibility of a particular parasite or other disease for much of the ill-health or morbidity in the people. One group claims that if malaria is eradicated, the capacity of the African will be vastly increased. Another group, especially the Egyptians, maintains that if only bilharziasis can be eliminated a healthy nation will be produced. Yet another considers that if the diet is balanced and adequate in quantity and quality the parasites can be disregarded and the problem of ill-health will be solved. Not one alone is correct. Although, in my opinion, the improvement of the nutrition is perhaps the most important single factor that helps to preserve a healthy population, one should not forget that the diet of the African has some advantage. Its low fat-content tends possibly to militate against degenerative heart disease. To obtain the best results, however, I believe we should approach the problem of disease from a wider aspect so that it is attacked from all angles at once.

THE CYCLE

The outlook for mankind exposed to these parasites is ultimately good. We can be hopeful because the parasites infect man only through his close contact with nature. The closer this association, the more likely he is to be infected with one or more of them. The parasitic diseases may thus be regarded as diseases of nature or environment, including the soil, the water, the vegetation, animals and insects. If in any of the diseases one can succeed in breaking a link in the chain of events, there is a good chance of avoiding the infection. Thus even if he lives in an endemic region, man can avert bilharziasis by avoiding contact with the waters. He need not contract hookworm as long as he wears shoes. If he lives in a mosquito-gauzed house, the cycle of the malaria parasite is affected and he stands a good chance of surviving the malarial season. Good drainage and spraying are also effective measures of breaking the cycle.

While there are many ways of preventing the disease I

think that perhaps the factor which most helped the White man in early settlements in Africa was his continuing to follow a European way of life and particularly his living in a European type of house. As long as his nights were spent in that house his chances of escaping the fever were good. In the small townships of Rhodesia and Nyasaland, at the turn of the century, few people died, but when they moved out to the country in the rainy season, even for a few days, they ran a grave risk of being cut off. The European settler who died of fever was not the townsman but the traveller, prospector, tramp, and transport rider. A very good example of the importance of this factor in the prevention of malaria is shown by the experience of the U.M.C.A. mission on Likoma island of Lake Nyasa in its early days. These brave missionaries believed that in order to convert the Natives they should live like them in the same type of huts and eating the same food. This experiment was a catastrophe. Their mortality was so terrifying that the London board had to intervene. This was largely through the efforts of Dr. Robert Laws, a medical man in charge of the Scottish Mission on the Lake shore nearby, whose men suffered far more lightly from malaria because they lived in brick houses. On the Scottish mission the mortality from the fever was negligible. When the U.M.C.A. missionaries changed their housing they too withstood the malaria.

Before 1898, when Ronald Ross proved the part played by the anophelene mosquito in the transmission of malaria, the outlook for European settlement in Africa seemed hopeless. The various governments, backed by the opinion of the medical profession, were loath to permit large-scale settlement on account of the fever. In fact tropical Africa was known as 'the White man's grave'. Except for the missionaries, even the few people who came out did not intend to settle and it was believed that the future of the White man in Africa could be 'counted in years rather than generations' (Felkin, 1895). Today the White man can live in any part of tropical Africa with little risk to himself or his family; provided he avoids close contact with the elements of nature the hazards of parasitic diseases can be overcome.

Finally we should remember that the tropical diseases of Africa are not static. Each forms a complex unit in which the parasite, the animal, man and his environment build up a chain or a whole. It is dynamic, evolutionary and ever changing and, as pointed out by Heisch in Kenya (1956) reacts with other wholes. At any time a disease may disappear or diminish, with man receding into the background and the disease confining itself to animals. Heisch has in fact evoked the theory of holism as propounded by that great South African statesman and philosopher, Jan Smuts, in order to explain the behaviour of some of these parasites.

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MOUTH INFECTION AND TREPONEMA VINCENTI

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It is well known that Vincent's spirochaete (and often the associated fusiform bacillus) is commonly found in smears from unhealthy gums and other mouth lesions, and sometimes from healthy mouths. The aetiological significance of the treponema in these conditions and of its commensal the fusiform bacillus is generally regarded as an unsettled question. Whatever its role may be, Vincent's spirochaete is commonly found in an inflammatory condition of the gums which may in severe cases go on to a gangrenous condition, laying bare both rows of teeth to the roots and leading to a toxic condition and even to a fatal issue.

MacCallum[1] mentions that, besides the mouth, Vincent's infection is found in the pharynx, bronchus and lungs. Park and Williams[2] state that it is found in gangrene and noma, and in the gum margins of dirty teeth. Bailey and Love[3] add acute superficial glossitis. Shera has recorded its presence in the rectum or sigmoid, where it causes a relapsing diarrhoea. The Extra Pharmacopoeia[4] refers to its presence in the eyes, nose and penis. Savill[5] states that these organisms may be found in ulcerative stomatitis, in acute septic scarlet fever, and in a form of acute tonsillitis. From my own observations I can add otitis to the list.

TWO CONSECUTIVE SERIES

To investigate the position in the African population of the Matatiele district of the Transkei, I examined the mouths of 224 consecutive patients. Of these, 141 (63%) showed clinical signs of gum infection. Smears were taken of each of these

and submitted to the Senior Government Pathologist, Durban, who reported that every one of the 141 smears (100%) showed the presence of Vincent's spirochaetes. These 141 patients were not necessarily complaining of mouth trouble. Indeed, only one in the series presented himself with a mouth complaint. Some were parents accompanying sick babies and others were school children and mine workers coming for routine examination.

In a second investigation 141 consecutive African patients were examined.* Of these, 10 were babies and 3 were toothless adults; smears were taken from the other 128 (89.4%), of which 126 (98.4%) were reported by the Government Pathologist as showing Vincent's spirochaetes. Of the 128 patients from whom smears were taken, 100 (78.2%) showed clinical signs of a gum infection (99 of these were positive for Vincent's) and 28 were from patients with clinically healthy mouths (27 of these were positive for Vincent's). The results of this investigation (other than those concerning the state of the teeth) are shown in Table I, which also includes the

TABLE I. SERIES OF 128 CASES

	Native		*Coloured*		*European*	
	Ratio	%	*Ratio*	%	*Ratio*	%
Infection present (clinical or laboratory)	127/128	99	48/50	96	*	
Vincent's positive	126/128	98	41/50	82	13/22	59
Vincent's negative	2/128	1·5	9/50	18	9/22	40·9
Healthy gums (no clinical or laboratory infection)	1/128	·75	2/50	4	*	
Clinically healthy mouth, Vincent's positive	27/128	21·8	6/50	12	*	
Clinical infection present	100/128	79	25/50	50	*	
Chronic or pyorrhoeal infection (clinical)	14/128	10·9	15/50	30	*	
Chronic or pyorrhoeal infection (clinical), Vincent's negative	0/128	0	3/50	6	*	
Chronic or pyorrhoeal infection (clinical), Vincent's positive	14/128	10·9	12/50	24	*	
Infected gums (clinical), Vincent's negative	1/128	·75	9/50	18	*	
Sub-acute on Chronic forms	*		2/50	4	*	
Diagnostic error (i.e. negative slides returned later as positive	†		2/50	4	3/22	14

The series of Natives and Coloured were consecutive, of Europeans as nearly consecutive as possible.
* Not noted. † Not noted (very small).

results of a similar investigation on Coloured patients (48 smears) and on European patients. These investigations show how widespread the clinical condition and the Vincent's infection are in the Matatiele district. Investigations in miners, industrial workers and school children in other places indicate that they may be common in Africans elsewhere in the Union. Coloured and European communities are also affected.

It would appear that the infection with *Treponema vincenti* mainly occurs in the mucous membranes, and that in severe cases it extends more deeply, sometimes resulting in gangrene.

* These were also investigated for dental conditions.

The alternative hypotheses are that this organism is the primary invader and the cause of the diseased condition; or that it is associated with some other primary invader (e.g., a virus), either as a virulent ally or as a harmless commensal. It is found in persons who appear to be in normal health and in what appear to be normal gums, and another theory is that it lies dormant (or as a saprophyte) to become active under certain conditions, such as malnutrition and ill-health. Possibly the gums become sensitive to its presence and react in a similar manner to an impetiginous eczema about a discharging ear. Whatever the role of this organism, it is important to recognize the gravity of the gum condition (and other diseases) with which it is associated, both in view of their extremely wide distribution and their severity in many cases.

SYMPTOMS AND SIGNS

The following are prominent symptoms and signs in this widespread condition of gingivitis. Sucking of the teeth or pressure on the gums (e.g. with a swab) will draw blood. This is sometimes observed in the absence of apparent inflammation. Spitting of blood after the use of a toothbrush is suggestive.

Taste. There is usually a foul taste of stale blood or pus. The taste of fresh blood also occurs, as well as a salty taste from the blood. The smell of the mouth depends on the severity of the disease, the habits of oral hygiene, the state of health, fever, etc. Africans do not generally complain of foul breath or bad tastes.

Pain. There is a dull neuralgic ache as a rule, which varies

in intensity. It is not often complained of. It is due to infected gums, carious teeth, infected jaw-bone, and loose teeth. Other forms of pain and throbbing also occur—from the pressure of chewing, contact with cold air or water, the eating of sweets, etc.; and from abscess formation. As the gums recede the exposed teeth are painful like the quick of a nail.

Oedematous gums, overlapping the teeth somewhat and producing pockets discharging mucoid pus, and flaps. This is more marked in the lower jaw. The gum is hyperaemic and red, according to the severity of the inflammation, but in some cases does not look very different from normal gum. The affected gum recedes to a certain extent with treatment, but never altogether recovers from its inflammatory state.

Receding gums are seen in older persons in whom the disease has persisted for many years. The oedematous soft tissue of the earlier years is replaced by fibrous tissue, which retracts and does not bleed so easily. This shrinking causes the gums to recede and the teeth become 'long' with 'windows' between.

VARIOUS FORMS OF THE DISEASE

No one appearance can be described as typical. Gums which appear to be healthy give positive smears as well as obviously inflamed gums. Where inflammatory process is present, the outer gum-margin is always more affected than the inner. Various gum states can be distinguished as follows:

1. *Apparently healthy gums*, clinically indistinguishable from the normal, but giving smears showing the Vincent organisms, which may be regarded as latent.

2. *Mild form.* A thin pink line is seen along the gum margin, or the gum may look normal, though it bleeds on pressure or on sucking. In a slightly more advanced stage this line becomes thicker and redder, sometimes presenting a striated appearance. Pain is slight or negligible, or there may be mild neuralgia. There is often satisfactory general health in spite of prolonged infection.

3. *Sub-acute oedematous form.* The inflammatory process is deeper and the gums redder and thicker (i.e., more oedematous), with flaps overlapping the teeth. The base of the gum becomes undermined and separated from the tooth, forming pockets, while the edge of the gums shows overgrowth of granulations (proud flesh). The infection does not extend to the rest of the mouth, though sometimes (not commonly) a tonsillar ulcer may occur, with sore throat. Africans usually do not complain of the pain unless it is very severe, and then they seek dental extraction rather than medical attention. Often they are only seen for some unrelated condition, e.g. dysmenorrhoea. If the infection becomes deeper a low-grade osteomyelitis of the jaw results in continuous pain, with some malaise and fever. Even in this state the patient may seek dental extraction rather than medical treatment. Sometimes the inflammation of the gum is more acute locally, about a stump or a carious tooth or tartar. The sub-acute inflammatory form is commoner than the acute toxic form (no. 6) and is usually found in young adults.

4. *Chronic*, or *pyorrhoeal*, or *fibro-purulent* form. This is a later stage, common in the elderly. The gums become more fibrous and less oedematous (not bleeding so readily) and recede, laying bare the teeth. The tooth bed is undermined and the pockets thus formed exude frank pus in place of the previous sticky mucoid discharge. The teeth eventually become loose. Mild pain is generally present. As a rule the patients enjoy fair health.

5. *Sub-acute on chronic* form. Less commonly acute inflammation supervenes on the foregoing (4). The gums become hyperaemic and bleed readily and the gum pockets literally drip mucoid pus, which runs down the side of the teeth.

6. *Acute toxic, inflammatory* and *gangrenous* form. This rarer form is observed in children only. The gums, tongue and mouth become inflamed and painful, bleeding readily, with oral ulcers associated. The gums sometimes become oedematous to the point of overlapping the teeth. Later a necrosis commences and a grey slough appears and, as the flesh decays, windows appear between the roots of the now

TABLE II. AGE INCIDENCE

Age (years)	*No.*	+	−	*H and* −	*I and* −	*H and* +	*I and* +	*H*	*I*
1-2* ..	14	2 (14%)	12	8	4	1	6	9	5 (36%)
3.. ..	12	11 (92%)	1	1	0	6	11	7	5 (42%)
4.. ..	12	9 (75%)	3	1	1	5	11	6	6 (50%)
5.. ..	12	11 (92%)	1	0	1	5	12	5	7 (57%)
6.. ..	12	12 (100%)	0	0	0	5	12	5	7 (57%)
7.. ..	12	11 (92%)	1	0	1	1	12	1	11 (92%)
8.. ..	12	8 (67%)	4	0	4	3	12	3	9 (75%)
9.. ..	10	9 (90%)	1	0	1	1	10	1	9 (75%)
10-12 ..	12	7 (57%)	5	2	3	2	10	4	8 (67%)
13-15 ..	12	10 (83%)	2	1	1	3	11	4	8 (67%)
16-19 ..	14	13 (93%)	1	0	1	5	14	5	7 (50%)
20-30 ..	12	9 (75%)	3	1	2	0	11	1	11 (92%)
31-40 ..	12	12 (100%)	0	0	0	1	12	1	11 (92%)
41-50 ..	12	10 (83%)	2	0	2	1	12	1	11 (92%)
60-75 ..	12	10 (83%)	2	0	2	1	12	1	11 (92%)
Total ..	182	144 (79%)	38	14	23	46	168	54	126 (69%)

No. = Number of patients.
\+ = Smear positive for Vincent's spirochaete.
− = Smear negative for Vincent's spirochaete.

H = Gums clinically healthy.
I = Gums clinically infected.
* 13-24 months.

loosened teeth. Fever, pain and loose teeth prevent eating. The child's condition, resembling that in cancrum oris, is highly toxic, with tachycardia, fever, sunken eyes, dehydration and exhaustion. Death may occur, but on treatment the child as a rule recovers rapidly. The teeth remain loose and fall out when the gums recede, and the gums remain in a chronic inflammatory state. The buds of the second dentition are not destroyed by the gangrene.

Age Incidence. Experience at Matatiele is at variance with Savill's statement[6] that 'gingivitis due to Vincent's spirochaete is common before the middle-age' and that of Bailey and Love[3] that it is essentially a disease of young adult life and it is exceptional to find it after the age of 35. In our patients the clinical condition is common at all ages and its percentage incidence tends to increase with age (Table II). Nevertheless the inflammatory process tends to be more active in younger people. If no teeth are present the organism as a rule is not found in the gums.

Tonsillar Infection. After gums the commonest site for Vincent's infection is the tonsils. In Table III are shown the results of the examination of smears taken from the tonsils of

TABLE III. EXAMINATION OF TONSILS FOR VINCENT'S SPIROCHAETE

	Total	*Vincent positive*	*Vincent negative*
Consecutive infants (age 3-10 months) without teeth	12	0	12
Consecutive infants (age 12-24 months) with teeth	12	1	12
Consecutive 'normal' tonsils	11	3	8
Consecutive acute tonsilitis (follicular membrane etc.)	17	1	16
Consecutive enlarged tonsils	11	4	7
Consecutive symptomatic sore-throats (N.A.D. on examination)	5	0	5
Consecutive cases without teeth and with tonsils present	10	1	9
Consecutive cases with teeth present and with tonsils absent (pharyngeal swab)	7	1	6
Case with no teeth and no tonsils (pharyngeal swab)	1	1	0
Total	86	12	74

86 patients in consecutive groups. In 12 cases Vincent's infection was reported (14%).

Wassermann Reaction. I have not found the Wassermann test to be an indication of infection with *Treponema vincenti.* For example in 11 related patients of good moral standards, whose Wassermann results were negative, the gum smears were all positive for Vincent's spirochaete. Similarly, 38 patients were tested for Wassermann. Of these 10 had clinical syphilis with positive Wassermann; 6 gave gum smears which were Vincent-positive and 4 Vincent-negative; in 2 the clinical diagnosis of syphilis was doubtful and the Wassermann reaction was negative; both these showed positive Vincent's infection. The remaining 26 were non-syphilitics with negative Wassermanns; 25 of them showed positive Vincent's infection and one (with inflamed gums, however), negative.

TREATMENT

Both from the preventive and curative point of view it is necessary to attend to the general health and to oral hygiene in the treatment of this widespread form of gingivitis.

In the African, nutrition is a factor of first importance, and the correction of malnutrition must take a high place in the combating of the disease. The question of diet is also involved in oral hygiene.

Loose and decayed teeth which are beyond repair should be extracted. A warning, however, should be sounded against the unnecessary extraction of teeth which are sound or can be repaired; so far as these are concerned treatment should be limited to the gums, and attention to the general health. Tartar deposits should be removed and also 'grit' and stains on the teeth. The tooth-brush should be soft. Hard tooth-brushes, especially when the gums are soft and friable, may do more harm than good. In some cases it is probably better merely to rinse the mouth. Tooth-pastes that are chemically irritant or unduly abrasive should be avoided.

Antibiotics. Aureomycin, given systemically, I find to be the most successful drug to subdue oral infection. The local application of aureomycin is less successful. Penicillin may also be used systemically and locally. A one-shot injection of 600,000 units is of great help in subduing acute oral inflammation, but in the toxic and gangrenous forms it is wiser to continue the treatment.

With antibiotic treatment the Vincent's infection, after disappearing, reappears in a few days. With aureomycin the disappearance tends to last somewhat longer. The gums never entirely heal under this treatment, but inflamed gums are not found to flare up again.

Sulphonamides. Sulphatriad has been found to improve the gum state, particularly in the more acute forms. Given with penicillin it produces a good response. The organism, however, returns and the gum never quite heals.

Mouth Lotions. Potassium chlorate may be used as a mouth wash or as a lozenge. It has a still more beneficial effect if taken orally, particularly in the more acute forms, especially when given with Sulphatriad, though the toxic or gangrenous forms respond quicker and more dramatically to penicillin. Hydrogen peroxide and mercurochrome are useful too. With these lotions, also, the gum never quite heals. Antibiotic lozenges have their use, but with long administration they make the gums and tongue sore.

Vitamins. Vitamin B_{12} and B complex, nicotinic acid and vitamin C have all been used, orally and by injection, with varying results. The improvement is usually limited.

Conclusion. The disease should only receive antibiotic treatment if the gum condition has got out of control and the infection has become acute. Antibiotics have taken the place of arsenicals, local and systemic, which the earlier text-books recommended.

SUMMARY

An account is given of an investigation into the occurrence of gingivitis and Vincent's infection in two series of cases

(chiefly Africans) at Matatiele, Transkei. Both the glossitis and Vincent's infection are shown to be extremely widespread.

A clinical account is given of the various forms of this condition.

The aetiology and treatment are discussed.

My thanks are due to Dr. M. C. Botha for his work, interest and encouragement, and to the members of his staff at the Government Pathological Laboratory, Durban.

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MANUAL REMOVAL OF THE PLACENTA WITHOUT GENERAL ANAESTHESIA*

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It has been generally accepted that manual removal of the placenta requires a general anaesthetic. However, it is proposed to show that this was found to be superfluous in a series of 102 cases. Anaesthesia carries its own inherent dangers and complications, with particular regard to manual removal. The procedure was safe and easy under a variety of circumstances and, on occasions, in all probability life-saving. No reference to placental removal without anaesthesia was found in the voluminous literature on the subject. The two main complications pertaining to retained placenta and manual removal in general are sepsis and shock, singly or jointly.

It is largely because of sepsis that teaching traditionally disapproves of manual placental removal. The more modern text-books, however, advise that the procedure today is safe, when performed where the proper indications exist, because of the strict asepsis, antiseptics and antibiotics now available. Macafee[1] can '. . . . state emphatically that it is a safe operation carried out at the right time and with due aseptic and antiseptic precautions.' There are many American papers[2-4] advising routine manual exploration (under anaesthesia) without an increased rate of sepsis or morbidity. Sepsis, therefore, need no longer be a deterrent factor, whether an anaesthetic is used or not.

SHOCK

In retained placenta there are two main principles with regard to shock both conforming with basic surgical tenets:

1. The best treatment is preventative.
2. If present, shock should, wherever possible, be corrected before manual removal is undertaken.

Shock in retained placenta may be due to: (1) Postpartum haemorrhage, (2) Credé's manoeuvre of placental expression, (3) the administration of anaesthesia or its complications, (4) complications of blood transfusions, (5) retained placenta.

1. *Postpartum Haemorrhage.* Macafee[1] states: 'Many generations of obstetricians have stressed the great danger of this procedure (manual removal) and have advised that it should only be employed *as a last resort*. This has, in the past, brought the operation into disrepute as it was quite true that it is dangerous when used as a last resort. It is now recognized that when carried out at the proper time it is not a dangerous or difficult operation and is definitely life-saving'. This confirms the surgical axiom of prevention of shock. However, the corollary is not stated, that manual removal can safely be performed forthwith without waiting for anaesthesia, i.e. can truly be a first and not a last resort. In Hagberg's recent survey of the University of Cape Town Flying Squad Service,[5] 60 of the 165 cases of postpartum haemorrhage had retained placenta, and a few more had completed the 3rd stage between the time of the call and the arrival of the squad. Two patients died with the placenta still in the uterus and almost all the others suffered from shock to a varying degree, with haemorrhage as the aetiological factor. This state of affairs need not develop if the placenta is safely removed in good time.

2. *Credé's Manoeuvre of Placental Expression.* Complete agreement has not been arrived at regarding the value of this procedure. It is stated to cause shock, and to be a procedure to be tried only once and never in the presence of shock. It is painful and often fails even in patients of less than average build; in my experience it is not a highly efficient or successful manoeuvre. In comparison, manual removal does not cause shock, is certain of success, is not often painful, and is not associated with a significant increased rate of sepsis. Hour-glass constriction is certainly handled more effectively *per vaginam* than abdominally.

3. *Anaesthesia and its Complications.* Dugald Baird's views[6] on general anaesthesia for manual removal are ' . . . that there is no obstetrical operation in which it is more desirable to have the assistance of an anaesthetist or colleague *skilled and experienced in giving anaesthetics*,' for the difficulties to be faced are legion even if a trained anaesthetist is in attendance at every birth as suggested by Macafee.[1] This in general is an impractical ideal; certainly anaesthesia is not invariably necessary, carrying with it all its inherent disadvantages, as follows:

(i) Unavoidable delay or difficulty in anaesthetizing.

(ii) Lack of premedication.

(iii) Induction vomiting, resulting from the presence of gastric contents.

(iv) Associated heaving, straining, restlessness, cyanosis, coughing, laryngeal spasm, or inhalation of acid gastric contents. These factors may make the manual removal a difficult and trying operation and are often enough to

* A paper delivered at the Obstetrical and Gynaecological Congress, Cape Town, April 1957.

precipitate a state of shock. Even ruptured uterus may occasionally result from manual removal under these circumstances.

(v) Anaesthetized cases need special nursing care and are often a source of anxiety for some hours.

(vi) Delayed effects, e.g. atalectasis or inhalation bronchopneumonia, may ensue.

(vii) Chloroform, especially, and ether and trilene prolong the 3rd stage and have a relaxing uterine effect.

4. *Blood Transfusion.* Macafee[1] states: 'The ideal to arrive at is that no patient should need blood transfusions. Manual removal at an early stage in treatment of postpartum haemorrhage will remove the necessity for many transfusions.' The dangers and complications of blood transfusion, even with compatible blood, include rigors and shock.

5. *Retained Placenta.* Sheehan[7] found that, in his series of almost 150 cases of maternal death with obstetrical shock, 30 had had retained placenta for 2 hours or more with little or no haemorrhage. He suggests manual removal after 1 hour as a routine procedure. Most writers agree on this; few placentae separate spontaneously after that time.

EVOLUTION OF MANUAL REMOVAL WITHOUT ANAESTHESIA

When on Flying Squad duty I regularly encountered heavily shocked patients with retained placenta and the full list of anaesthetic troubles. The patients had usually been given tea etc. before the squad arrived; there was generally only an inexperienced anaesthetist available, or none at all. These cases were all given morphine, ¼ gr. intravenously, as part of the treatment for shock as suggested by Mayer.[8] One day severe haemorrhage forced me to remove the placenta manually without anaesthetic. The ease and speed of the procedure and absence of complications prompted the present series of manual placental removals, carried out under varying circumstances by different registrars and house-surgeons, skilled and unskilled.

BASIC TECHNIQUE

After treatment of shock, when necessary, the patient was, in all cases, left in the dorsal position and catheterized. It is generally agreed that movement may precipitate a drop in the blood pressure,[8] and, in this series, the dorsal position was as effective as the lithotomy one.

Manual removal was effected by the usual bimanual technique, the hand always being reinserted even if the placenta and membranes were apparently intact on inspection, since the latter is not always reliable.

Blood-pressure and pulse readings were taken on arrival (Flying Squad cases) or on admission (hospital cases), immediately before and immediately after manual removal, and again 30 minutes later, for comparative studies. The patient's opinion was also recorded both just after and 30 minutes after manual removal. In this way objective and subjective views were both obtained.

It was not found necessary to wait for the blood pressure to rise even to 100 mm. Hg systolic in shocked cases before effecting removal, so long as it was definitely rising on anti-shock treatment—a point of some importance where haemorrhage recurs as the blood pressure is being restored (Case 1).

Case 1

Age 30. *Parity* 8. A Flying Squad case.

Indications for Removal: Haemorrhage 80 oz. +. Retention 190 minutes.

Treatment before Removal: Blood Transfusion, 3½ pints; morphine, ¼ gr. I.V. 40 minutes before.

Condition: On arrival, B.P. 0/0 and pulse not felt. Immediately before removal, B.P. 80/30 mm. Hg and pulse 140 per minute. Immediately after removal, 90/50 and 128. 30 minutes later, 130/90 and 105.

Patient's Remarks. Immediately after removal, 'Much easier than childbirth.' 30 minutes later, 'Feels much better.'

Total Quantity of Blood given: 4 pints.

Ergometrine administered before removal was not regarded as a contra-indication. It is not generally accepted that this increases the frequency of hour-glass constriction rings, as borne out by Flew[9] *et al.* who, in 500 consecutive cases in which ergometrine was given before the completion of the 3rd stage, had met no case of hour-glass constriction nor any case in which manual removal was required. In our series hour-glass constriction *per se* did not prove a major difficulty in manual removal without anaesthesia. It was treated by amyl nitrite inhalation under a towel placed over the face (case 2).

Case 2

Age 17. *Parity* 1. A Flying Squad case.

Indications for Removal. Haemorrhage: ± 42 oz. Retention 220 minutes.

Treatment before Removal. Blood Transfusion 1½ pints. Morphine, ¼ gr. I.V. 20 minutes before.

Condition: On arrival, B.P. 70/45 mm. Hg and pulse 140 per minute. Immediately before removal, 95/65 and 132. Immediately after removal, 115/75 and 100; hour-glass ring; amyl nitrite. 30 minutes later, 105/70 and 120.

Patient's Remarks. Immediately after removal, 'Abdomen feeling better. It was not sore.' 30 minutes later, 'I.V. drip needle hurt more than removal.'

Total Quantity of Blood given: 2 pints.

No morphine or other sedation is necessary to prevent shock if the patient's condition is good (case 3). This is illustrated by the 17 cases in the control group (table I).

Case 3

Age 30. *Parity* 1. Not a Flying Squad Case.

Indications for Removal. Haemorrhage 26 oz. Retention lasted some minutes. Difficult forceps delivery under local anaesthetic, followed by haemorrhage.

Treatment before Removal. Blood transfusion, *nil*; morphine, *nil*.

Condition. On admission, B.P. 110/80 mg. Hg and pulse 96 per minute. Immediately before removal, 140/80 and 100. Immediately after removal, 130/80 and 110. 30 minutes later, 120/80 and 76.

Patient's Remarks. Immediately after removal, 'Not painful.' 30 minutes later, 'As before.'

Total Quantity of Blood given. Nil.

Manual removal effected in the manner described, must perforce be gentle, and rupture of the uterus should not occur as a result of the procedure.

Refinements of Basic Technique

Intravenous Morphine, though not part of the basic technique, was of great value in shocked patients to allay the associated restlessness and anxiety which form a neurogenic basis for shock.[8] When given subcutaneously in profound shock it may fail to be absorbed because of the poor peripheral circulation. Adams[10] states that intravenous morphine in therapeutic doses is a very safe, very potent hypnotic and analgesic which also depresses the attention and weakens the impression of external stimuli. The blood pressure is

essentially unchanged, although respiration is depressed to some extent in some cases. It is particularly valuable where '. . . one desires to obtain the effects quickly, and it is very useful in cases in which a short trying procedure has to be borne.' It is suggested that a well diluted dose of 1/6 gr. should first be slowly administered, to eliminate sensitivity and overdosing. In our cases ¼ gr. undiluted was slowly administered intravenously in the 78 cases which were given morphine—all successfully. This is a big dose and, although it proved to be quite safe, it is felt that 1/6 gr. should probably suffice. No fatal case is quoted under these circumstances in the British and American pharmacopoeias.

Intravenous pethidine is held by most authorities to cause at least as much respiratory depression as morphine, to cause a definite drop in blood pressure (rare and minimal with morphine), to give more sensitivity reactions, and to produce considerably less analgesia, dose for dose, than morphine. It was therefore not used.

Trilene *via* the self-inhaler mask was an additional safe and useful feature in occasional cases. In this way minimal intermittent doses only were administered.

Daptazole, the antidote to the respiratory depression of morphine, was never indicated, nor Lephidrone, even in severely shocked cases.

ANALYSIS OF SERIES

The series reported here constitutes 102 fully recorded cases, of which 72 were multiparous (15 in their 7th or more pregnancy). The cases were divided into 4 groups, the first being the basic control group, which had no sedation whatsoever at any stage. In addition the procedure was used in nearly 40 more cases (without any failures), which are not included because of incomplete records (Table I).

TABLE I. MANUAL REMOVAL WITHOUT ANAESTHESIA. ANALYSIS OF SERIES OF 102 CASES

Group	*Description*	*No. of Cases*	*Sedation*	*No. with Hour-glass Constriction*	*Comment and Reason for Removal*
I	Hospital Cases	12	0	1	Retention or PPH
	(Control Group)	5	0	0	Prophylactic after forceps under local anaesthetic
II	Flying Squad Cases	34	Intravenous Morphine	10	Very shocked—25, Less shocked—5, Not shocked—4, Retention 1-4 hours Haemorrhage gross in all cases
III	Hospital Cases	38	Intravenous Morphine	18	Retention 5 min.-2 hrs. Haemorrhage up to 70 oz.
		7	Already heavily sedated	0	Eclamptics 2, Pre-eclamptics 5. 20 of these cases had trilene self-administered
IV	Hospital Cases	6	Morphine	0	Prophylactic removal after forceps delivery under local anaesthetic

Sedated cases: 85 (with 28 hour-glass constrictions). In 78 of these i.v. morphine was given.
Non-sedated cases: 17 (with 1 hour-glass constriction).
Total: 102 (with 29 hour-glass constrictions).

COMPLICATIONS

1. *Hour-glass constriction ring* developed in 29 cases. It is not a serious matter and always yielded to amyl nitrite and steady gentle separation and traction. It was often diagnosed in advance by the physical signs of separation without expression. In 2 hour-glass cases in the series manual removal was carried out without difficulty after transfer to hospital following failure on the part of the intern to effect manual removal in the patient's home.

2. *Morbidity.* Only the 68 hospital cases could be followed up. 20 had to be placed on antibiotics on account of prolonged labour or repeated vaginal examinations. Antibiotics were not given as a routine measure. Two patients were definitely ill and another 4 ran high temperatures (9%), which is not considered excessive for this type of case.

3. *Apprehension.* The patients invariably cooperated by relaxing and breathing deeply, sometimes with the aid of the Trilene mask. Some of the control group were very apprehensive, yet removal was easily effected. In no case did much difficulty arise or failure occur because of apprehension on the part of the patient.

4. *Significant Pain.* In 72 cases adequate follow-up notes on this point are available: 6 patients stated that the pain was worse than that of childbirth, which was the criterion selected for the purpose of comparison (see Case 4), 8 said it was approximately the same, and in the other 58 (even in the control group where morphine was not given) it was appreciably less. Even where the manoeuvre was very painful and difficult, there was no evidence of shock.

Case 4

Age 34. *Parity* 4. Not a Flying Squad case.

Indications for Removal. Haemorrhage 40 oz. Retention 70 minutes.

Treatment before Removal. Morphine, ¼ gr. I.V. 6 minutes before. No blood transfusion.

Condition. On admission, B.P. 130/85 mm. Hg. Pulse 80 per minute. Immediately before removal, 150/100 and 85. Immediately after removal, 150/95 and 90 (placenta extremely adherent). 30 minutes later, 130/80 and 90.

No blood transfusion given in hospital.

Patient's Remarks. Immediately after removal, 'Very painful; more so than childbirth.' 30 minutes later, 'Not at all pleased.'

Careful analysis confirms the basic safety and ease of the manual removal procedure.

CONCLUSIONS

A series of 102 cases of manual removal without anaesthesia under varying circumstances is presented with illustrating case records.

1. It is considered that the method is easy and non-shocking even in inexpert hands; is not unduly liable to lead to sepsis under modern circumstances and may be of life-saving significance in some cases. Basic surgical principles should be observed, especially that shock is better prevented than treated, which principle confirms the value of the method.

2. Intravenous morphine is suggested as a safe and valuable refinement in the procedure. Trilene given by the standard self-inhaler mask is another successful refinement.

3. A case is made out in favour of omitting the anaesthetic when manual removal is practised, thus avoiding the frequent undesirable sequaelae and dependence on an anaesthetist, who may not be available at the time of need. The omission of the anaesthetic converts a relatively major operation into a relatively minor one.

SAMEVATTING

'n Reeks van 102 gevalle van manuele verwydering van die plasenta onder wissellende omstandighede en sonder algemene narkose word aangebied, met sorgvuldige ontleding van die gevalle ter stawing van die stelling dat narkose vir dié doel onnodig is, indien nie byna altyd ongewens nie. Die volgende gevolgtrekkinge word bereik:

(1) Die prosedure blyk veilig, maklik en sonder skok (vergelyk hier Credé se manoeuvre) te wees selfs al sou die verloskundige ongeoefend wees in plasentale verwydering. Dit kan lewensreddend wees en is nie gepaard met noemenswaardige voorkoms van infeksie nie, maar sjirurgiese grondbeginsels moet gehuldig word, o.a. dat dit beter is om skok te verhoed as te behandel—welke feit die waarde van die prosedure beklemtoon.

(2) Binneaarse Morfiene is 'n veilige hulpmiddel beide as verdowings- en skokmiddel. Die Trilene-masker waardeur die pasiënt haarself van gas bedien, verskaf verdere verdowing indien nodig.

(3) Daar word daarop gewys dat narkose in plasentale verwydering waarskynlik skielik en onverwags is en die pasiënt onvoorbereid, en dus ook vol gevare en komplikasies, al sou die narkotiseur ervare en beskikbaar wees. Deur plasentale verwydering sonder narkose uit te voer, word tydverspilling, moontlike skok en komplikasies van die narkose grotendeels uitgeskakel.

I wish to thank Prof. James T. Louw for his constant encouragement, advice and criticism, and my registrar colleagues, the house surgeons and the nursing staffs of the hospitals for their invaluable aid, especially in dealing with patients in the University of Cape Town Flying Squad calls. Thanks are also due to the Superintendents of the Peninsula Maternity, St. Monica's and Mowbray Maternity Hospitals for permission to publish.

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A TANK RESPIRATOR CONSTRUCTED IN CAPE TOWN

T. B. McMURRAY, M.Ch. Orth. (L'pool), F.R.C.S. (Edin.)

Cape Town

During the recent poliomyelitis epidemic, there has been a scarcity of mechanical respirators. A large number of the patients, particularly in the younger age groups, are suffering from either weak intercostals or from interference with the diaphragm, and it has been necessary to assist their breathing in the initial and recovery stages. During the infective period, all patients have been treated at the City Hospital where a number of respirators are available, and all those who have required continuous treatment by mechanical means, have been retained in that hospital until they can continue outside a respirator for long periods.

In other hospitals where active treatment has been carried on, difficulties have arisen because many of these patients, while being able to survive outside mechanical respirators, have still considerable weakness of their intercostals, and this, together with the colder weather, makes them peculiarly liable to embarrassment from naso-pharyngeal or respiratory infections. As a prophylactic against serious chest complications, it is necessary that these patients be treated intermittently in the respirator in order to provide a full aeration of their lungs and avoid serious complications.

It is realized that obviously a large number of additional respirators cannot be made available at short notice, and, moreover, the cost, which is in the nature of £1,200 to £1,400, makes this economically difficult. For this reason, the possibility was investigated as to whether a Tank Respirator fulfilling all the requirements of the imported model could be manufactured and supplied in Cape Town.

With this in view, I approached Mr. J. Consani of Consani's Engineering Works, and put the problem to him with some suggestions as to how such an Iron Lung might be constructed. He told me that never before had his factory undertaken such work, but he was willing to try and with co-operation of those doctors dealing with polio cases, he felt that a satisfactory solution might be found.

After some 6 weeks' work, the first iron lung was produced, and tested at the Conradie Hospital. The original model was designed for children under the age of 5, and although

mechanically sound and able to carry out its function satisfactorily, certain minor improvements were necessary. The first model was then returned to the factory and the second model was produced.

The present lung is able to take a patient of up to 6 feet 3 inches tall, and it has been tested and compared within the last few weeks and found to be completely satisfactory.

1. *The Body of the Lung*

The body of the lung is constructed of 1/8 inch steel plate. This material is used because although it is heavy, it is also extremely strong and much easier to work than dur-

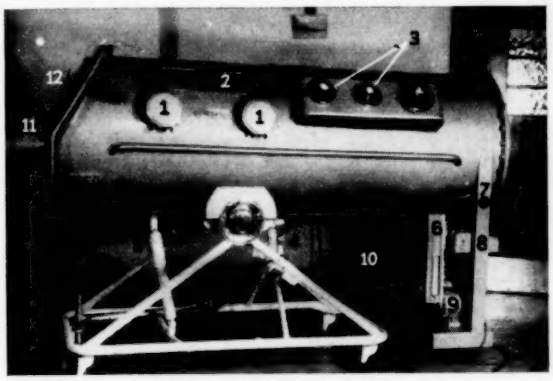

Fig. 1

1. Hand-holes, two on each side.
2. Inspection aperture covered with perspex, one on each side.
3. Inlet and outlet valves with variable pressure.
4. Jack for tilting the lung.
5. Hydraulic shock absorber to control the tilt.
6. U Tube to act as gauge for pressure.
7. Switch for lighting and heating.
8. Switch for electric motor.
9. ¼ Horse-power electric motor.
10. Wire guard covering motor and gear box.
11. Head rest.
12. Clamp to secure rubber valve air lock.

alumin or any of the light alloys. The problem of moving the lung from place to place is not a great one in South Africa where we have an ample supply of manual labour to assist us.

II. *The Pump*

I have always felt that a pump constructed separately from the lung is unnecessary; it certainly makes for difficulty in moving the lung about and is much more easily damaged.

Secondly, the tube through which the air is sucked in and out of the lung gives rise to draughts and also increases the dead space, a factor which must be seriously considered when dealing with a child. An increase in the amount of air to be sucked in and out of the lung assumes paramount importance when the body of the patient is small, because the elasticity of air and the relative diminution of suction necessitates an increase in the excursion of the pump in order to secure proper pressure. Any increase in the relative amount of air space in the lung or in the tube, together with a natural resilience, tends to diminish the suction power. For this reason, the pumping mechanism is placed directly

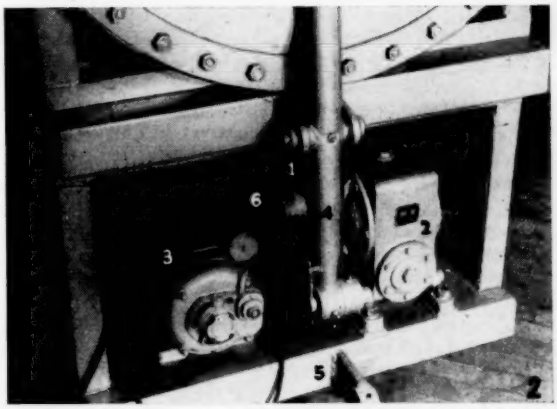

Fig. 2. Motor Unit

1. Driving wheel perforated in three places giving rise to different excursions of the driving arm.
2. Gear box.
3. ¼ Horse-power electric motor.
4. Driving arm.
5. Bracket to secure emergency hand apparatus.
6. Pulley belt.

at one end of the lung. The body of the lung is cylindrical in shape and is covered at the pumping end by a sheet of ¼ inch rubber; the centre of this rubber sheet is connected by large washers to a driving arm which moves to and fro and draws the rubber sheet in and out of the cylinder, so that the patient lies virtually within the body of the pump.

The question has arisen as to how long this rubber sheet will last, being under intermittent tension? The size of the large washers which are both inside and outside of the sheet vary, so that the same amount of tension is not applied on each side. This diminishes the wear and tear on the rubber sheet.

The Motor

The motor is an ordinary ¼ horse power electric motor, which is connected first by pulley to a reduction gear which enables the pump to work at various speeds either 12, 16, or 22 excursions per minute. The reduction gear drives a steel wheel which is perforated at various levels to receive the connection link of the driving arm of the pump.

The reason for this is that the excursion of the pump can be varied to a considerable extent, so as to ensure that when a small patient is placed within the pump, adequate pressure can be achieved, particularly in suction.

The Stretcher

At the other end of the pump there is an aperture for the head with two small arms protruding, so that a sling can support the head outside the pump.

The stretcher inside the pump is on ball bearings and rollers, and is connected with the opening end of the pump, so that when the spring clips are released, this end of the respirator, together with the stretcher, can be drawn out to about 4½ feet.

The air lock is secured in the conventional manner to the head, opening by means of metal clamps, and consists of the usual sponge rubber neck piece with zip fasteners, although latterly we have been experimenting with an oblique fitting

collar which seems to be much more satisfactory since it is variable in size. (Normally there have to be five or six sizes of the sponge rubber collar in order to secure a satisfactory air lock for all patients.)

Closure of the door is effected by 6 spring fasteners and the rubber collar ensures that an adequate air lock is produced.

Observation and Nursing

There are two perspex windows placed one on either side of the body of the pump, and below them, about 18 inches from the head end, are two arm holes on each side, all equipped with spring quick-release locks. Rubber valves inside these locks prevent the air excursion, when it is necessary for the nurse to put her hands inside the pump in order to treat the patient. The provision of two hand holes on either side ensures that two nurses each looking through the relative perspex window are able to administer the patient at one time.

The Control of Negative and Positive Pressure

The control of negative and positive pressure is achieved by means of two cone valves placed on the side of the pump. By this means both the positive and negative pressures can be individually controlled and their fine adjustment made possible. In addition, a U tube containing oil is fixed to the side of the pump which gives an accurate reading of the pressures. In testing this lung, it was shown that a far greater control of both negative and positive phases can be achieved in this lung than with most of the imported models.

Tilting

Often in nursing patients in an iron lung, it is necessary to tilt the lung itself in order that mucous and other respiratory exudates do not run down into the bronchi. The tilting mechanism is produced by means of a jack similar to a car jack placed under the lung, together with an hydraulic shock-absorber which allows the lung to be tilted to nearly 45°. This tilting is carried out smoothly and slowly; in addition, the inner side of the door of the lung is equipped with rubber pads so that the patient's shoulders are not pressed too hard against the door.

Mobility

The solid framework of this lung rests on 4–4½ inch rubber wheels which allows it to be wheeled about within the ward or from ward to ward. It is however, rather heavier than the conventional Iron Lung, but it is not impossible for it to be lifted on to a truck or lorry for transportation. Its heaviness is compensated for by the fact that it is of extremely strong construction and will stand a fair amount of knocking about.

Emergency Power

As with all machinery which relies upon electrical motors for power, the lung is subject to breakdown, and this may prove serious if the power is temporily cut off. For this reason, an emergency handle has been provided which takes about 15 to 20 seconds to slip into place, so that the lung may be worked by hand. It is found that one nurse can carry this out quite easily without exhaustion for about 10 or 15 minutes, but relays of orderlies would be able to carry on keeping the lung working for an indefinite period.

SUMMARY

This iron lung has the following advantages:

1. *The Price:* The price of this lung is less than that of the imported models.
2. *Construction:* It is constructed entirely in South Africa and all materials are purchased in the Union except the electric motor.
3. *Resistance to Damage:* The extremely solid construction makes it unlikely to be damaged by any normal accident.
4. *Control:* It is extremely easy to control and to work in both elements of pressure.
5. *Emergencies:* In emergencies the lung may be completely worked by hand.

I should like to thank Mr. J. Consani for his work in the production of this lung and for the many ideas and for the tremendous enthusiasm which he has displayed in this work.

IMPRESSIONS OF A SHORT TOUR IN SOUTH AFRICA

C. A. BOZMAN, *O.B.E.*

Director, Wellcome Museum of Medical Science, London. Formerly Director of Public Health, Burma

I came to South Africa at the invitation of the South African Medical Association to attend the Medical Congress in Durban and to show something of the type of display to be found at the Wellcome Museum of Medical Science in London.

A few words on the organization behind the Museum may not be out of place here, for the story is an unusual one. It began in 1880, when two young Americans set up in England a manufacturing pharmaceutical firm under the style of Burroughs Wellcome & Co. This venture quickly prospered and, on the early death of Burroughs, became the sole property of the surviving partner, Henry Wellcome. From the start Wellcome devoted a part of the profits earned by his business to research and he soon showed a remarkable flair for picking for this work men of the highest calibre. Among the many workers in the Wellcome Research Laboratories who have made outstanding contributions to medical problems the names of Henry Dale, Wenyon, and Hoare, are known and respected internationally.

As his affairs prospered and became established, Wellcome developed his personal interest in the history of medicine and pharmacy and archaeology. He succeeded in collecting a library of books and medical incunabula which is unrivalled, and material illustrating medical and pharmacological practice throughout the ages, which also is unique. Eventually he combined the manufacturing firm of Burroughs Wellcome with its subsidiaries and the Museums, Library and Tropical Medicine Laboratory referred to above, into one organization, the Wellcome Foundation Ltd. On his death all the shares in the Foundation devolved on a Trust, and when the Foundation has met the costs of the pharmaceutical manufacturing firms and the costs of maintaining the Museums, the Library and the Tropical Medicine Laboratory, all the money that remains is paid to the Trust. That body distributes what it gets in the way of dividends as gifts to further research into medical and allied problems in accordance with the terms of Wellcome's will.

The Wellcome Museum of Medical Science has been in existence for 44 years. It presents written accounts of diseases at post-graduate level and supplements the script with visual aids such as photographs, photomicrographs, X-rays and charts as well as pathological and entomological specimens. Many thousands of doctors and medical auxilliaries visit the Museum annually.

The particular display chosen for the Medical Congress was a duplicate of the section on Bilharziasis. This collection was

selected for a variety of reasons. The condition is important in the Union, as well as other parts of Africa, South America, the Middle East and the Far East, affecting a total of some 120 million of the world's population. Notable contributions towards the problems of aetiology, epidemiology and control have been made and are still being made by workers in the Union and the Rhodesias. While the broad principles that underlie the successful control of this infestation are well known, the practical difficulties in their application are such that bilharziasis remains one of the major unsolved health problems of the areas in which it is rife. It was with interest that I heard both at the Congress and afterwards the views of those who are dealing with this problem in the Union Health Department, and of workers in laboratories under the control of the South African Institute for Medical Research, the Council for Scientific and Industrial Research, and the various South African medical schools. Later I was able to visit a field control experiment at Nelspruit, and saw some of the practical difficulties that arise in an experimental control area where work has been in progress for some time.

That 'doctors differ' is an oft quoted truism, and during my stay in the Union I heard many different views on this particular problem. This in itself is, of course, as immaterial as it is, perhaps, inevitable. What is most material and most evident, was the enthusiasm and drive I found in all I met and their refusal to be put off by difficulties and temporary failure. I should like to say here that it is this spirit of enthusiasm that has struck me so forcibly in the many and varied medical activities that I have been privileged to see during my visit to South Africa.

In an article like this it is not possible to comment on all I saw during my 6 weeks' stay; the attempt would probably only be boring to the reader. I would like, however, to refer briefly to a few of the activities I have seen which have been of particular personal interest to me. First among these are the arrangements for medical education in the five medical schools in South Africa. The Natal University Medical Faculty is concerned at present with the training of Native and Coloured students. Here the curriculum followed presents certain unusual features that make this experiment one of considerable interest. The thought and care and spirit of high endeavour with which the staff at the Durban Medical School have approached their task are all immediately apparent to the visitor. The standards of the first graduates from this school will be observed by many with great interest, for it is possible that the medical faculty of Natal University have pioneered an approach to medical education that other schools in other countries will want to copy. Two of the remaining schools admit both Europeans and Natives, and two are restricted to Europeans only. While these are on orthodox lines, in all of them the visitor meets with the same spirit of energy and drive already referred to. To be a medical student in South Africa must be a most stimulating and, perhaps, exhausting experience!

The Union health authorities face a formidable task in providing for the large Native population efficient medical services for which the direct financial contribution paid by the Native is negligible. I had two interesting and informative visits to a large Native hospital just outside Johannesburg. This must be a model for work of this kind. Cleanliness, order and efficiency were everywhere apparent and this, in a hospital that trains its own Native nursing staff, reaches a degree that in a wide experience of hospitals of this type I have never seen equalled. There is here a wealth of clinical material which makes one envy the staff and the students who attend as housemen from the near-by Witwatersrand medical school.

At Westford I saw in operation a scheme for the control of leprosy. South Africa employs a technique that is, I think, unique inasmuch as leprosy is a compulsory notifiable disease. I was assured that, contrary to what is held elsewhere, this actually works and, in practice, the number of 'hidden cases' is believed to be small. I felt that a possible contributory factor to this is the success that attends the scheme of treatment the Superintendent employs for his early cases. He is achieving results that compare most favourably with those I have seen in other regions.

Through the courtesy of the Director and the Deputy Director I was able to see something of the wide scope of the work carried out by the staff of the South African Institute for Medical Research. Here valuable contributions are being made towards the solution of problems of nutrition, of cancer incidence and of cardiac and circulatory failure, to mention only those subjects in which I happen myself to be specially interested.

Until one actually sees this country it is hard to appreciate the vast spaces of the Union. The problem of bringing hygiene and reasonable curative services to the scattered European farmers and the African population in the kraals and reserves poses many difficulties. The need for many trained European and Native medical auxiliaries supported by a scheme of health education by radio, cinema and school services soon becomes apparent. Such may be already in existence or planned for the near future; if so, and if tackled with the same 'vim' that is apparent in the large urban centres in which perforce I spent most of my time, success would seem to be assured.

I leave South Africa hoping that I shall be able to meet in London at the Wellcome Museum of Medical Science some of those who have shown me so much kindness and consideration during my visit to this energetic and stimulating country.

GREETINGS FROM SCOTLAND

We are enabled to publish the text of the speech in which Professor Norman M. Dott, C.B.E., of Edinburgh, read messages from the Scottish Royal Colleges and Medical Schools, at the banquet of the South African Medical Congress in the Durban Town Hall on 17 September 1957, as follows:

'My first duty and great pleasure is to bring you greetings from Scotland. The Royal College of Surgeons of Edinburgh send you this message, signed in the name of the College by Walter Mercer, President:

' "We, the President and Fellows of the Royal College of Surgeons of Edinburgh, the oldest Surgical Corporation of the British Commonwealth of Nations, now in her 452nd year, offer our warmest congratulations to those meeting to celebrate the 41st South African Medical Congress. Your efforts and achievements in the cause of Medicine in South Africa are a source of pride to your country and to ours. And so, to this important meeting we send our fraternal salutations." '

Professor Dott proceeded: 'The royal College of Physicians of Edinburgh through its proper Officers, desires me to associate it with this message of greetings to you.

'Likewise the Royal Faculty of Physicians and Surgeons of Glasgow directs me to salute you in the name of its President, Professor Stanley Alstead, and its Fellows.

'The Scottish medical schools of Aberdeen, St. Andrews, Edinburgh and Glasgow desire me to express their cordial good wishes and to bear their greeting to this great meeting of the profession in South Africa. They cherish the warmest sentiments towards you and they anticipate a most successful Congress here.

'I feel that I can also speak for my professional colleagues generally in Scotland—hardly a family but has some cherished memory contact or relationship with South Africa. They wish you well in the important activities of this Congress.

'I feel that I can also speak for the people of Scotland in this matter. Their feeling for you is warm and cordial and their interest in you is lively and affectionate, as those of you who have sojourned with us well know.

'I find it much more difficult to express my personal appreciation of your kindness to me—for I am quite overwhelmed by it. Starting at the present with this magnificent feast, which I am appreciating highly in respect of good food, good drink and good companionship, I look to the recent past; South Africa has provided me in Edinburgh with some of my most helpful, most loveable and ablest colleagues, who now surround me with their kindness. I look a little further back and recollect that South Africa supported and encouraged my earlier professional efforts in no small measure. Let me include in this not only her medical profession, but also her courageous patients, who adventured overseas and entrusted their brains to me. And a little further back I see the charming and inspiring group of South African

students whom I accompanied through medical school in Edinburgh.

'When I turn to the future—the immediate future—I am a little awe-struck by the lecturing prospect before me, but it is so seasoned with high honour and kind interest that its forbidding aspect is changed to smiles. In any event any repayment I can make for what I owe South Africa now and in the past is but slight and trivial. And of the more distant future—it appears as through rose-coloured spectacles. For I know that here in South Africa the interests and ideals that we cherish in Scotland will be promoted, that what we may have helped to start in former days will be improved upon, and that we shall look to South Africa with confidence for help, encouragement and inspiration.

'Two weeks since I took ship across the Minch from South Harris in the Hebrides. It was a lovely day. The island hills gradually faded in the atmosphere that is of the mystery of Scotland—sea, hill and sky clouded in a dim mystic vision—etherialized. Yet at a thought I could see again in sharp clarity the burns, the lochs and the heather blooms. So I think it is here with many of you who know Scotland—a dim memory of the past, but at a thought revivified on such an occasion as this when old friends meet.

'Perhaps it is the same for your memories of Edinburgh Royal Infirmary—a dim outline of grey stone with its pinnacles. But at a thought the intimate scene at the front gate—the house surgeon, perhaps yourself, seeing off his chief—was it Alexis Thomson, Caird, Stiles, Wilkie, Fraser?

'To those who have not yet visited Scotland, I would urge that time and space these days are at your disposal—23 hours now will bring you to a warm welcome.

'That reminds me that Scotland still has something to give. Those new Britannia Aircraft are excellent, relatively silent the advertisements say. We took off from London Airport and I overheard this conversation of three South African passengers returning home:

'*No.* 1 *sitting at the window seat.* "Is that Wembley?"

'*No.* 2. "No! It's Thursday."

'*No.* 3. "So am I. Lets have a Scotch and Soda."

'They did so forthwith.'

QUESTIONS ANSWERED

Question:

1. Is it generally acknowledged as proven that aspirin is an irritant to the gastro-intestinal tract, and may cause bleeding?

2. Do its modifications such as adding calcium carbonate and citric acid, or magnesium carbonate and aluminium glycinate, to make it neutral and soluble, not greatly diminish its analgesic properties?

3. Has phenacetin any deleterious effect on the blood and, if so, what are the signs and symptoms?

Answer:

Gastroscopic investigations have shown that aspirin in contact with the gastric mucosa does produce an inflammatory action, even to the extent of haemorrhage. Aspirin tablets should be crushed before they are swallowed, in order to avoid large particles lying undissolved which, while breaking up slowly, may produce local erosion, especially on an empty stomach. Calcium aspirin is soluble in water, but is unstable. Calcium acetylsalicylate tablets contain aspirin and calcium carbonate, usually with citric acid. The effect is identical with that of acetylsalicylic acid but absorption is probably more rapid.

Phenacetin may produce cyanosis from the formation of methaemoglobin, but this is rare.

ANGLO AMERICAN MEDICAL REPORT

Dr. J. H. G. van Blommestein's 'Medical Consultant's Report' to the Anglo American Corporation of South Africa Ltd., for 1956 has been published as a 63-page document consisting chiefly of tables. It covers the administration of 18 hospitals for Natives containing 2,735 beds. This Mining Group employs 60 full-time and 5 part-time medical officers, as well as 11 health and antimalarial officers. These medical services cover certain gold mines on the Reef and in the O.F.S., colleries in the Union and Southern Rhodesia, copper mines in Northern Rhodesia, and diamond fields in the Union and South West Africa.

A considerable amount of work and research has been devoted to the design and planning of the post-war hospitals in the Group and to remodelling and reconditioning the older hospitals. 'Apart from the basic requirements of any hospital, one of the major problems encountered in the design of the newer hospitals is to make them conform to the most modern requirements and, at the same time, make them sufficiently flexible not only to meet immediate needs and conditions, but to be capable of extension and alteration in order to meet future requirements. As far as the design of the new Group hospitals is concerned, it is felt that this difficult task has been accomplished.' Although no expense has been spared in installing essential equipment and providing essential facilities, by adopting plain, simple features of design and eliminating all architectural 'frills', the cost per bed in Group hospitals has been lowered by as much as 50% compared with other modern establishments of similar size. Hospital kitchens, adds Dr. van Blommestein, have been designed to meet the specific needs of Natives, and Native patients have a free choice of the foods to which they are normally accustomed.

One of the interesting developments during 1956 was the universal introduction of a miniature, 75 mm. film, as a substitute for large film in chest radiography. The use of this film, which is one of the provisions of the regulations pertaining to the Pneumoconiosis Act of 1956, was foreshadowed by the Group's medical department as far back as 1952 when it was decided to introduce miniature film in the equipment at the Group's Orange Free State gold mines, as it was considered more frequent recourse to X-ray examinations would be a valuable aid in combating the incidence of two of the most important diseases occurring in mining, pneumoconiosis and pulmonary tuberculosis.

'The value of this procedure is clearly demonstrated when a comparison is made of the incidence rates of these two diseases occurring among Reef gold mines workers (where mass miniature chest radiography was not available) with those occurring among the workers in the Orange Free State. No doubt, cognisance was taken of these results which had already been obtained by the use of such apparatus in the Orange Free State and at the Witwatersrand Native Labour Association Depot, when the regulations under the new Act were framed.'

An average of nearly 70,000 Natives were employed on the 13 Group gold mines on the Reef and in the Orange Free State in 1956. In all, there were 159 deaths from mining accidents. The lost shift rate, in respect of accidents, was 1·275 a Native per year on Reef mines and 2·806 in the Orange Free State. The greatest number of accidents was caused by falls of hanging—the major cause—and by rock, trucks and locomotives.

Although some of the mines in the Anglo American Corporation Group come under the classification of 'hot mines', there were only 9 cases of heatstroke (including 1 death) recorded last year. When it is remembered that some thousands of Native mineworkers are employed by the Corporation in conditions of excessive heat and humidity, this figure is an indication that measures employed for prevention of heatstroke are effective.

Incidence rates for disease, accident and other causes within the Group are highest in the gold mines of the Reef and Orange Free State and lowest on the diamond mines. One explanation

of the difference in rates is that environmental conditions play an important role in affecting the health of employees. Conditions of temperature and humidity are generally normal on the diamond mines, where half the employees are working on alluvial diggings, and the remainder in shallow mines where there is practically no variation between surface and underground temperatures. The copper mines of Northern Rhodesia are somewhat deeper and temperatures on the whole higher; and in the Reef and Orange Free State gold mines environmental conditions underground are decidedly above normal.

Although the incidence rates for disease and accidents on the gold mines were higher than elsewhere, they may nevertheless be considered good. 'If the lost shift (or absentee) rate for disease is taken as an index', states Dr. van Blommestein, 'it will be seen that in 1956 the lost shifts per Native per annum were only 2·428 on the Reef gold mines—which is a commendable figure, particularly when it is compared with absentee rates for various industries employing all types of workers throughout the world.'

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

'Positol'

'Positol' (Sitosterols, Lilly) represents an entirely new concept for reducing elevated serum cholesterol. The manufacturers supply the following information:

'Positol' is a suspension of sitosterols, principally beta-sitosterol, for oral administration. The sitosterols are sterols that are limited to the plant kingdom. Chemically, they are very similar to cholesterol, the beta form differing from cholesterol only by the presence of an ethyl group.

Indications. 'Positol' is indicated in hypercholesteraemia, regardless of aetiology. Chronic elevation of serum cholesterol is commonly seen in patients with coronary atherosclerosis (angina pectoris, myocardial infarction), cerebrovascular disease, xanthomatosis, nephrosis, uncontrolled diabetes mellitus, and hypothyroidism. It may be used as an adjunct to such specific therapy as is available, e.g. thyroid substance in hypothyroidism and insulin in diabetes mellitus.

Patients with inherent defects in cholesterol metabolism (e.g. essential hypercholesteraemia) may be expected to show only small reductions, since 'Positol' would not alter the basic defect. 'Positol' may cause a partial reduction in such cases by preventing the absorption of dietary and biliary cholesterol.

Side-effects and Contra-indications. There are no contra-indications, nor have side-effects been observed. Only a very small quantity of 'Positol' is absorbed, and it is rapidly eliminated. After 3 years of clinical use, 'Positol' therapy has been shown to be devoid of any harmful effects.

Dosage. The usual dosage is 1 tablespoonful (approximately 15 c.c.) *immediately before* each meal of medium size. The dose should be increased to 1½ or 2 tablespoonfuls when large or high-fat meals are consumed. A total daily dose of 4-6 oz. may be required in some individuals. For maximum therapeutic effect, any additional food taken between meals should be preceded by a whole dose or fraction thereof. The patient should be impressed with the necessity for taking a proper dose of 'Positol' before every meal.

As a guide to therapy, it is suggested that serum total-cholesterol levels be determined initially and at 2-4 week intervals until the dosage is established that will produce the maximum effect. A downward trend of the serum cholesterol level is often noted at the end of 1 week, with further decline during the succeeding 4 weeks. Occasionally, maximum results require therapy for as long as 2 months.

Supplied. Suspension 'Positol' is supplied in bottles of 1 pint.

Manufacturer: Eli Lilly & Company, Indianapolis, USA.

Enquiries: P.O. Box 7605, Johannesburg, or from your Lilly representative.

* * *

Mysteclin-V Capsules

Squibb Laboratories (Pty.) Ltd. announce this new phosphate complex of tetracycline for faster and higher initial tetracycline blood levels for antimicrobial therapy plus added protection against monilial superinfection, and supply the following information:

Mysteclin-V contains a tetracycline phosphate complex which has an exceptionally wide range of antimicrobial activity against pathogenic organisms, including gram-positive and gram-negative bacteria, certain large viruses, certain rickettsias and *Endamoeba histolytica*; and also a Mycostatin component which provides specific activity against *Candida* (*Monilia*) *albicans*.

Advantages. Mysteclin-V, besides providing faster and higher tetracycline blood levels, also prevents the fungal overgrowth, particularly moniliasis in the intestinal tract, which is frequently seen when the bacterial equilibrium is disturbed by the administration of broad-spectrum antibiotics. When this overgrowth becomes pronounced, it may give rise to clinical moniliasis, including vaginitis, anogenital pruritus and thrush. Severe and even fatal monilial infections following antibiotic therapy have been reported with increasing frequency. The tetracycline phosphate complex in Mysteclin-V does not contain any sodium metaphosphate; therefore the clinician will have no problem with added sodium intake. It is a well-tolerated antibiotic, well absorbed, producing high blood, urinary and cerebrospinal levels in a short period of time, and diffusing readily into body tissues to combat the infection. Mycostatin is also particularly well tolerated. Side-effects or allergic reactions have been encountered very rarely during the widespread use of Mycostatin.

Indications. Mysteclin is indicated for many common infections, including those of the respiratory, gastro-intestinal and genito-urinary systems which are amenable to antibiotic therapy. Because of its wide range of anti-microbial activity, Mysteclin-V is particularly useful in the treatment of mixed infections. It is especially useful for patients who are most likely to be susceptible to the overgrowth of *Candida albicans*. Among such patients are those requiring high or prolonged antibiotic dosage, debilitated or elderly patients, diabetics, infants (particularly prematures), patients receiving concomitant cortisone or related steroid therapy, patients who have developed a monilial complication on a previous broad spectrum therapy, and women particularly during pregnancy.

Dosage. The suggested minimum dosage is one capsule 4 times daily. Higher dosages, such as 2 capsules 4 times daily may be required for severe infections or for those who do not respond to the smaller dose. Paediatric dosage should be calculated upon the tetracycline content so as to supply 10-20 mg. per lb. body-weight each day, in divided doses, according to the type and severity of the infection. Treatment for all patients should generally continue for 24-48 hours after symptoms and fever subside. It is advisable to treat streptococcal infections for a full 10 days to prevent the development of rheumatic fever. Even more prolonged therapy may be necessary for subacute bacterial endocarditis and certain staphylococcal infections.

Supply. Mysteclin-V capsules, each containing tetracycline phosphate complex equivalent to 250 mg. of tetracycline hydrochloride, and 250,000 units of Mycostatin, are available in bottles of 12 and 100.

Further information and samples for clinical evaluation are obtainable from Squibb Laboratories (Pty.) Ltd., 80 Jorissen Street, Braamfontein, Johannesburg.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. C. W. Coplans, of Sam Newman House, Burg Street, Cape Town, Specialist in Physical Medicine, who has been away owing to indisposition, will resume his practice on 18 November 1957.

* * *

Vth Congress of the International Society of Internal Medicine. The preliminary programme has been published of this congress, which is to be held in Philadelphia, USA, on 23-26 April 1958. Enquiries should be addressed to the Secretary-General, 4200 Pine Street, Philadelphia 4, Pa, USA. Members planning to attend the congress must complete the prescribed registration form and send it before 1 February 1958. The annual session of the American College of Physicians will be held at Atlantic City, USA, from 28 April to 2 May, and members of the congress are invited to attend. A tour has been arranged starting from Atlantic City on 7 May; the itinerary includes Washington, Baltimore, Ann Arbor, Niagara Falls and Boston, and will terminate on 15 May at New York.

* * *

Professor E. L. Bortz, M.D., of the University of Pennsylvania, Philadelphia, USA, who took a prominent and most acceptable part in the proceedings of the South African Medical Congress recently held at Durban, left for New York by air on 26 September. The following is an extract from a letter he has since written to a member of the Association:

'With the remarkable experiences of the last three weeks we find ourselves still travelling from place to place. I must say that in all of my travels in previous years, we have never experienced the gracious hospitality and kindly friendship which you wonderful people in your great nation extended to us from the States. We were much impressed with the high quality of research work in the various centres and, of course, it was a unique privilege to appear on the program of the Congress with your distinguished teachers and Sir Russell Brain and Sir Harry Platt.'

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Union of South Africa. Department of Health. Notification of formidable epidemic diseases and poliomyelitis in the Union during the period 18 October-24 October 1957:

	Poliomyelitis				
	Eur.	*Nat.*	*Col.*	*As.*	*Total*
Transvaal	3	2	–	–	5
Cape Province ..	2	2	–	–	4
Orange Free State ..	–	–	–	–	–
Natal	1	–	–	2	3
Totals	6	4	–	2	12

Plague, Smallpox, Typhus Fever: Nil.

Corrections

Two (2) Native cases (Lady Frere District) notified in News Letter No. 39 of 1957 have subsequently been diagnosed as not poliomyelitis. One (1) European case (Pinetown District) notified in News Letter No. 41 of 1957 has subsequently been diagnosed as not poliomyelitis.

REVIEWS OF BOOKS : BOEKRESENSIES

THE INTERNATIONAL GEOPHYSICAL YEAR

The International Geophysical Year. By Werner Beudeler. 71 Pages. $0.60, 3s., 150 fr. Paris: Unesco. 1957.

Contents: Section I. The Earth as a Planet. *Section II.* The Science of Geophysics. *Section III.* The International Geophysical Year. *Section IV.* The Scope of the International Geophysical Year. *Section V.* The Exploration of the Atmosphere. *Section VI.* At the Border of the Universe. *Section VII.* Prelude to Space Flight. *Section VIII.* Probing into the Sun's Mysteries. *Section IX.* Exploring the Earth. *Section X.* A Strange Land—Antarctica. *Section XI.* Value of the Programme. Glossary.

The international physical year has now begun. It includes scientific observations in every civilized country of the world and expeditions to the Antarctic by several nations. It has brought together in cooperation countries not usually on friendly terms. The newspapers and broadcast stations of the world have publicized various aspects of the I.G.Y. to so great an extent that everyone has heard of it and no man of professional status cares to remain ignorant of its main aims or the means taken to achieve them.

The author has written a simple and easily read account of the development of the idea of the I.G.Y. from earlier and successful, though less ambitious, attempts at scientific cooperation between nations—the first and second polar years. He describes the organization responsible for the arrangements and shows that the scientific observations are of common interest to mankind. He describes the main branches of Geophysics, the physics of the Earth and its atmosphere, and how the study of the Earth naturally expands so as to include observations of cosmic rays and of the radiation and physics of the sun. In a booklet of this small size it would be unreasonable to expect much space to be devoted to South Africa's contribution to the I.G.Y., but a good deal of attention is devoted to the scientific exploration of the Antarctic, which concerns South Africa very closely.

An interesting description, well illustrated, is given of the artificial satellites and of the manner of their launching by means of multiple rockets. He discusses the use of rockets for exploration of the upper atmosphere and shows the value of observations of the sun's ultra-violet radiation and of solar flares. In short, the author describes the main activities of the I.G.Y. and discusses their importance. The booklet discusses these matters in such a way that, although scientifically sound, it is easily followed by the non-expert and should be of interest to every intelligent person.

W.S.

CORRESPONDENCE : BRIEWERUBRIEK

FULL-TIME MEDICAL OFFICERS AND LIFE-INSURANCE EXAMINATIONS

To the Editor: I should like to refer to the letter under the nom-de-plume *Jaap* which appears on page 1080 of the *South African Medical Journal* issued on 19 October 1957, and to state that, as far as this Department is concerned, no full-time medical officer is at liberty to engage in private practice without prior approval from this office in terms of section 24 of the Public Service Act No. 54 of 1957; and should he do so he renders himself liable to a charge of misconduct in terms of the above Act.

This permission will only be considered in very special circumstances.

In regard to local authorities where medical officers in full-time employment are subsidized by this Department, such subsidy will be withdrawn if they engage in private practice without the prior consent of this Department.

J. du Pré le Roux
Secretary for Health

Department of Health
P.O. Box 386
Pretoria
28 October 1957